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PHYSICAL DIAGNOSIS

PHYSICAL DIAGNOSIS

BY

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WITH THREE HUNDRED THIRTY-SEVEN ILLUSTRATIONS

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THIS BOOK IS DEDICATED TO

DR. GEORGE DOCK

TO WHOM EVERYONE WHO HAS HAD A PART IN THE MAKING OF THIS BOOK IS INDEBTED

28390

PREFACE

For some time I have considered writing a book on physical diagnosis which would embody the work covered in a course on the technic of physical examination and normal physical diagnosis and a course on abnormal physical diagnosis.

When the publishers requested me to revise Dr. Rose's work, I consented, provided they would allow me to rearrange and rewrite the subject matter as seemed necessary to carry out my ideas. To this they consented, and the result appears on the following pages.

Much of Dr. Rose's excellent work has been retained, but a rearrangement has been effected, so that the book is now divided into two parts.

Part I is devoted to subject matter which has been found useful in the course on technic of physical examination and normal physical diagnosis. I have endeavored to cover each diagnostic procedure before taking up another. For instance, the inspection of the entire body is discussed before palpation is introduced. In practice this plan may not always be advisable, but students, in my experience, have a better working technic when this plan is followed.

In addition to the work covered in the text, demonstrations by specialists on ear, nose, throat and eye diseases are recommended, as their clinics are especially equipped and students are stimulated by receiving instructions in special technic from specialists. A demonstration on electrocardiography, where students can see the mechanism of the electrocardiograph, is recommended, and a demonstration on radiography and fluoroscopy is of value to familiarize the students with the operations of x-ray apparatus.

To check the accuracy of inspection and percussion, fluoroscopic examinations of subjects whose Litten's phenomenon and cardiac dullness have been outlined with lead strips, is recommended.

The numerous references to pathologic conditions in Part I are intended to emphasize the importance of the procedure under discussion and to stimulate the curiosity of the student. The technic of eliciting those physical signs which are produced only in pathologic conditions is discussed in Part I so that the student can begin his examination at the bedside with a fair working knowledge of physical signs, as well as the ability to make a routine physical examination.

At least three complete physical examinations of normal subjects, supervised by competent instructors, should be completed in connection with the textbook work.

A chapter on electrocardiography with normal electrocardiograms by Dr. Drew Luten, and a chapter on the principles of x-ray diagnostic procedures with examples of normal radiograms by Dr. Sherwood Moore, have been added to Part I.

Part II deals with the physical diagnosis of disease. As in most other works on the subject, most of the space is devoted to diseases of the thorax, as covered in the usual course on physical diagnosis. While one feels that a course in applied physical diagnosis should cover the entire body, present teaching arrangements in most medical schools make this impossible. It, therefore, seems best to leave the application of the technic not covered in the second part of this work to teachers and works on general medicine and the specialities.

The text should be used as a supplement to clinic and bedside demonstration. Illustrations from autopsy material are also recommended.

Dr. Luten has contributed a new chapter on cardiac arrhythmias, and has contributed various paragraphs on electrocardiographic diagnosis throughout this section of the work. Dr. Moore has edited the x-ray diagnosis scattered throughout this section.

New illustrations, including many radiograms, have been added. Several new chapters and parts of chapters have been added to make the work more useful as a textbook.

I desire to express my sincere thanks to Dr. Sherwood Moore and Dr. Drew Luten for their contributions to this work; to Dr. Sidney Schwab for suggestions and criticisms of the section on sensory phenomena and reflexes; to Dr. David Barr and Dr. G. O. Broun for their suggestions and criticisms; and to the Department of Radiology of the Washington University School of Medicine and the Missouri Pacific Hospital for radiographs and photographs.

To Miss Dorothy L. Hollman I wish to express my sincere appreciation for her invaluable aid in the preparation of this volume.

WARREN P. ELMER.

St. Louis, Missouri.

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PHYSICAL DIAGNOSIS

PART I

THE TECHNIC OF PHYSICAL EXAMINATION AND PHYSICAL EXAMINATION OF THE NORMAL BODY

SECTION I

CLINICAL ANATOMY AND PHYSIOLOGY

The clinical anatomy and physiology of the head, neck, and extremities should be sufficiently familiar to second year medical students for purposes of physical diagnosis. A brief review of the clinical anatomy and physiology of the pulmonary, circulatory, and abdominal organs, however, is essential for a thorough understanding of physical diagnostic signs, both in the normal and diseased body.

CHAPTER I

CLINICAL ANATOMY AND PHYSIOLOGY OF THE THORAX

The bony thorax resembles in shape a truncated cone with an anteroposterior flattening, which causes the transverse diameter to exceed the anteroposterior diameter by one-fourth. When the thorax is viewed in the living subject, however, the upper portion, which corresponds to the apex of the cone which is formed by the bony thorax, exceeds in transverse diameter the lower portion as a consequence of the investment of the superior portion of the thoracic cage by the bones and muscles of the shoulder girdle.

The walls of the thorax are formed by the rigid vertebral column posteriorly, and by the ribs posteriorly, laterally, and anteriorly, invested by muscles, subcutaneous tissues and integument. The sternum enters into the formation of a large portion of the anterior wall; and posteriorly the thorax is reinforced by

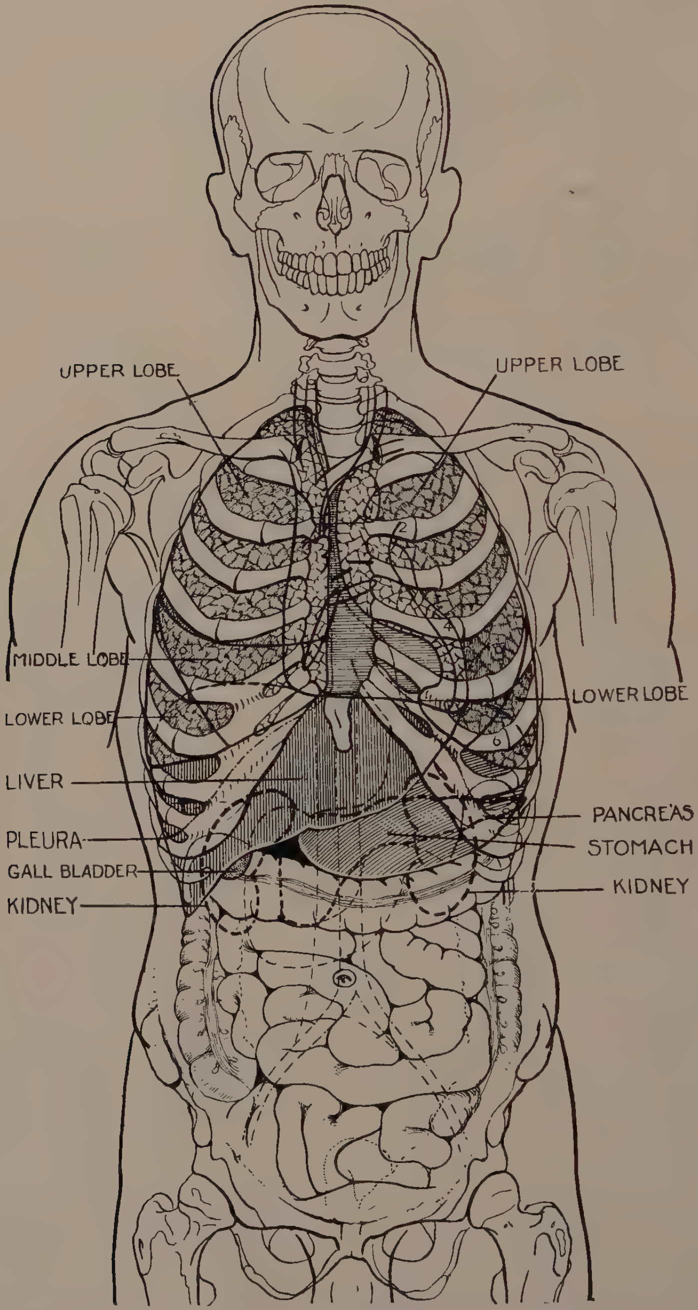

UPPER LOBE
UPPER LOBE
MIDDLE LOBE
LOWER LOBE
LOWER LOBE
LIVER
PANCREAS
PLEURA
STOMACH
GALL BLADDER
KIDNEY
KIDNEY
2
4
6

Fig. 1.—Relations and surface markings of thoracic and abdominal viscera. (Anterior view.)

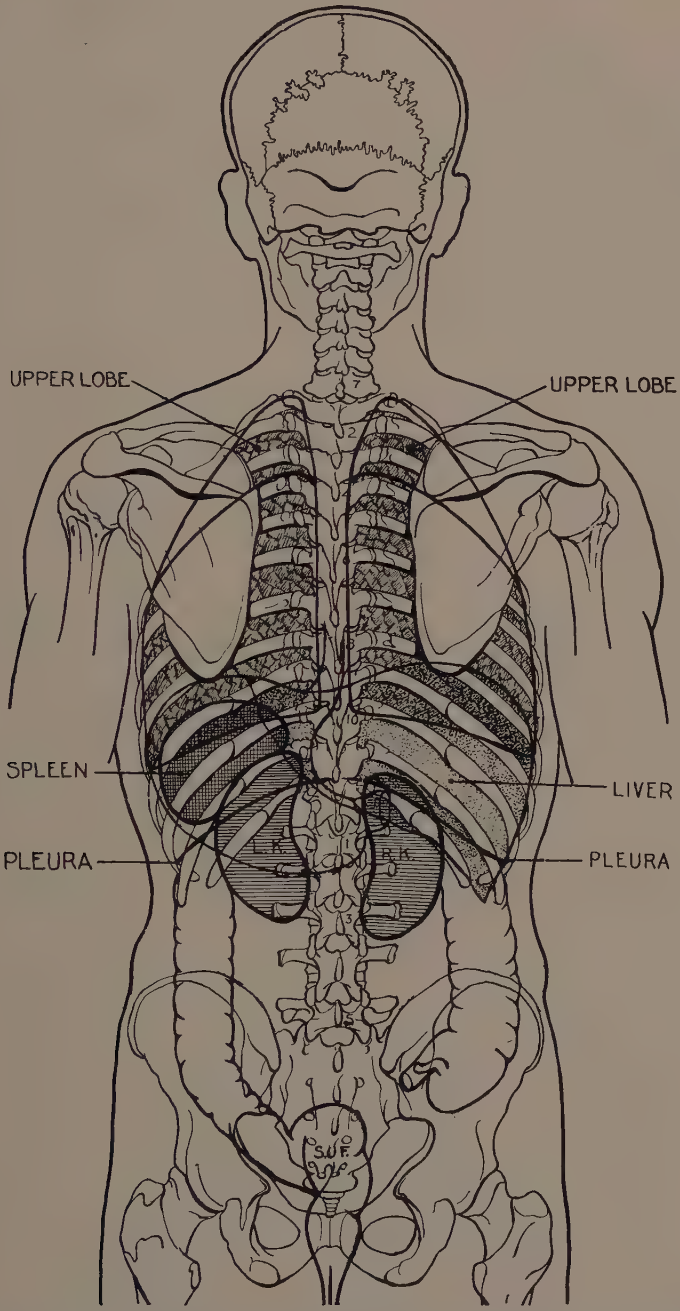

UPPER LOBE
UPPER LOBE
7
2
8
10
SPLEEN
LIVER
L.K.
R.K.
1
PLEURA
PLEURA
3
S.U.F.

Fig. 2.—Relations and surface markings of thoracic and abdominal viscera. (Posterior view.)

the superjacent scapulæ, which cover the ribs upon either side of the vertebral column from the level of the second to the seventh thoracic vertebra.

The intercostal spaces are occupied by the intercostal muscles, which are further reinforced by the parietal pleura, investing fascia, and the integument. These spaces are traversed by the intercostal vessels and nerves, the latter sending out their lateral cutaneous branches in series at points situated midway between the spines of the thoracic vertebræ and the sternum. Similarly, the terminal filaments of the intercostal nerves emerge at points in series near the lateral margins of the sternum. In the presence of intercostal neuralgia the points of exit of these cutaneous nerves are not infrequently subject to pain upon pressure, constituting Valleix's points of tenderness.

The *thoracic wall* varies in thickness in different regions of the chest, and also with the degree of physical development of the individual. Clothed in all parts by the overlying musculature, it is further reinforced posteriorly by the interposition of the osseous scapulæ, and anteriorly by the mammary gland in the female subject. In the axillary, infraaxillary, interscapular, and subscapular regions, on the other hand, the thoracic wall does not attain great thickness; consequently in these regions physical signs arising within the thorax are more clearly transmitted to the examining ear. In the subject of excessive muscular development, as well as in the presence of general obesity, a thick chest wall is the rule, while in the poorly nourished individual with imperfect development of the muscular system, the walls of the thorax are unduly thin and the scapulæ are readily displaced from their normal beds. The thoracic wall possesses less thickness in the female subject than in the male, owing to the greater degree of muscular development in the latter class. In the child, again, the chest wall is very thin and resilient, and physical signs arising within the thorax are transmitted thence with a corresponding increase of intensity.

The thoracic wall of the normal subject possesses an inherent elasticity, yielding readily to variations of pressure exerted from without or from within the cavity of the thorax. Consequently, characteristic deformity of the contour of the thorax readily results from occupation or from disease as, for example, the funnel-breast deformity of the shoemaker or carpenter, the barrel-chest of hypertrophic emphysema, or the elongated, alar thorax of chronic phthisis. As a result of this great plasticity of the thoracic parietes, careful inspection of the contour of the thorax assumes a very important rôle in physical diagnosis.

The thoracic cavity, containing the essential organs of respiration and circulation, is limited inferiorly by the diaphragm and superiorly by the dome of the parietal pleura, which is reinforced in this region by a thickening of the investing fascia, Sibson's fascia. The capacity of the thoracic cavity is not commensurate with the external dimensions of the thorax. Inferiorly the dome of the diaphragm during expiration ascends as high as the upper border of the fifth rib in the right midclavicular line, and to the lower border of the same rib in the corresponding line upon the opposite side of the thorax; but, as if to compensate in some degree for this deficiency inferiorly, the cavity ascends beneath the dome of the pleura into the root of the neck to a height varying from one to one and one-half inches above the clavicle.

The position of the diaphragm, depending as it does upon the relative pressures in the thoracic and abdominal cavities, is a very variable one. Fluoroscopic studies have demonstrated that the position of this important muscle varies with the posture of the patient and with various pathologic conditions arising in the thoracic and abdominal cavities. The diaphragm occupies the highest position when the subject assumes the dorsal decubitus, and in this position it exhibits the most extensive excursion during tranquil respiration. In the standing posture the muscle occupies a distinctly lower position and its excursions are less marked, while in the sitting position the dome falls still lower and the respiratory excursions during tranquil respiration are less extensive than in the recumbent or standing posture.

In the normal subject the two halves of the diaphragm are seldom symmetrical, and their relative excursions are rarely equal. Moreover, in the presence of extensive excavation, fibrosis or collapse of one lung, attended by compensatory enlargement of its fellow, the diaphragm is elevated upon the side of the diseased lung and is depressed upon the side of the emphysematous organ. Such a change in the position of the diaphragm is commonly attended by displacement of the mediastinal structures, as pointed out in a subsequent paragraph. Similarly, unilateral variations in intraabdominal pressure attending subphrenic abscess, and hepatic or splenic enlargement are likewise attended by elevation and relative fixation of one side of the diaphragm. A general increase of intraabdominal pressure causes the entire muscle to occupy an abnormally high level, whereas in the presence of a bilateral pleural effusion the diaphragm occupies an unduly low position.

In the normal subject at the completion of expiration the lateral portions of the diaphragm are in contact with the thoracic walls from the seventh to the tenth ribs. When, during inspiration the muscle becomes separated from the chest wall, a distinct undulation may frequently be noted if the patient is exposed to oblique illumination. Occasionally during expiration, as the diaphragm in its ascent again adapts itself to the thoracic wall, a similar undulation may be noted in the same area. This phenomenon, when it is demonstrable, was shown by Litten to be an indication of adequate diaphragmatic excursion.

The thoracic cavity is divided by reflections of the parietal pleura into three chambers; namely, a *pleural cavity* upon either side of the median line, intervening between which is a third cavity, the *mediastinum.*

The *pleural cavities,* occupying the lateral regions of the thoracic cavity, extend upward into the base of the neck to the extent of one to one and one-half inches above the clavicle, and downward as low as the attachment of the diaphragm to the thoracic walls. The vertical diameter of each pleural cavity is diminished by the ascent of the diaphragm during expiration, whereas the transverse and anteroposterior diameters of the cavity are increased by the excursion of the chest wall during inspiration.

The pleural cavity is lined internally by a thin serous membrane, the pleura, which closely invests its walls, in which situation it is termed the *parietal pleura.* For purposes of description and accurate localization, several subdivisions of the parietal pleura are recognized according to the distribution of the membrane. The portion of the membrane which invests the anterior, lateral, and posterior walls of the cavity constitutes the *costal pleura;* the portion which clothes the superior surface of the diaphragm, forming the inferior limit of the pleural cavity, is designated the *diaphragmatic pleura;* while the folds which are reflected from the anterior to the posterior thoracic walls, forming in this wise the lateral walls of the mediastinum, are termed the *mediastinal pleura.* The same membrane is reflected from the mediastinal wall on to the lung at its root, clothing the external surface of the lung completely and dipping into the fissures of the organ, forming the *visceral pleura.*

In the normal subject the pleural membrane is moistened with a small amount of serous fluid, which permits the visceral and the parietal pleura to glide noiselessly over each other during the movements of the lung and the chest wall during respiration. In the presence of inflammation of the membrane, however, the pleural surface loses its smooth, glistening appearance, and becomes coated with a variable amount of fibrinous exudate, giving rise to a coarse or grating sound during the respiratory movements, which is recognized by the clinician as the pleural friction sound.

In the normal subject the pleural cavity has a negative pressure approximating 6 mm. of mercury, by virtue of which the elastic lungs are closely applied to the parietal pleura as air enters the lungs during inspiration. Indeed, it is largely due to the inherent elasticity of the pulmonary parenchyma and the balance maintained between the negative pressure obtaining in the pleural sac and the positive pressure in the air passages that the heart and mediastinal structures are maintained in their normal position. When the pleural cavity is opened by a penetrating wound of the thorax or by the establishment of a bronchial fistula, this delicate balance is disturbed and mediastinal shifting ensues, with displacement of the heart and varying grades of shock.

The *mediastinum*, the central portion of the thoracic cavity intervening between the pleural cavities, is limited anteriorly by the sternum and costal cartilages, and posteriorly by the bodies of the thoracic vertebræ, while its lateral walls are formed by the reflection of the parietal pleura from the anterior to the posterior wall of the thorax. This important portion of the thoracic cavity is arbitrarily divided into four subdivisions; namely, the superior, posterior, anterior, and middle mediastina.

The *superior mediastinum* is represented by the portion of the mediastinal space which is situated above the lower border of the manubrium sterni anteriorly and the lower border of the body of the fourth dorsal vertebra posteriorly. As in the case of the remaining subdivisions of the mediastinum, its lateral walls are formed by the mediastinal reflections of the parietal pleura. This space contains the intrathoracic portion of the trachea together with the tracheal glands, the esophagus, and the superior portion of the thoracic duct. The superior mediastinum also lodges the arch of the aorta, with the initial portions of the three great arterial trunks which spring from its convexity; and here also are found the innominate veins, uniting to form by their confluence the superior vena cava.

The *posterior mediastinum* represents the downward continuation of the posterior portion of the superior mediastinum, whence it extends as low as the upper surface of the diaphragm, which separates it from the abdominal cavity. Bounded laterally by the mediastinal pleura and posteriorly by the bodies of the dorsal vertebræ, the posterior mediastinum is in direct relation anteriorly with the pericardium and heart. The posterior mediastinum contains the descending thoracic aorta, the esophagus in close relation with the pneumogastric nerves, a portion of the thoracic duct, and likewise a portion of the azygos veins. The space also lodges a chain of lymphatic glands, which are apt to become enlarged in the presence of intrathoracic malignant disease.

The *middle mediastinum* is situated immediately below the anterior portion of the superior mediastinum, and it is interposed between the anterior mediastinum ventrally and the posterior mediastinum dorsally, while its inferior limit or floor is formed by the central tendon of the diaphragm. That this is the most important subdivision of the mediastinum to the student of physical diagnosis is evident, when it is recalled that this limited space contains the pericardium and heart, the ascending aorta, the pulmonary artery with its two primary branches, the bifurcation of the trachea, the primary bronchi with their related bronchial glands, as well as the lower portion of the superior vena cava and its junction with the azygos veins.

The *anterior mediastinum* is a narrow space situated immediately behind the sternum. It is limited posteriorly by the pericardium and laterally by reflections of the parietal pleura from the anterior thoracic wall. The space is of little diagnostic interest, save that it lodges a few lymphatic glands, which may become enlarged or become the seat of malignant disease.

As the mediastinum is traversed by such a variety of important structures, it is evident that physical signs arising from mediastinal disease are apt in many instances to manifest themselves in the form of pressure symptoms. Pressure generated within this space, being exerted upon the large blood vessels, upon the large lymphatic tract represented by the thoracic duct, upon the esophagus or upon the air passages, will in each instance produce physical signs of definite localizing value. Moreover, as a result of the close contiguity of so many vital anatomic structures within a restricted space, inflammation of a single structure is apt to involve adjacent organs, resulting in the formation of inflammatory adhesions or frank abscess formation.

As a result of the elasticity of the lateral walls of the mediastinum, this subdivision of the thoracic cavity with its contents readily suffers displacement in a lateral direction in response to variations of pressure in the adjacent pleural cavity. Extensive induration or collapse of a lung is attended by displacement of the mediastinum toward the side of the lesion, and is usually associated with compensatory enlargement of the sound lung and characteristic alterations in the position of the diaphragm. Similarly, the mediastinum may be displaced by the traction of pleural adhesions, or by the pressure of an accumulation of fluid or gas in one pleural cavity. Thus, in the presence of an extensive pleural effusion, the shifting of the mediastinal structures occasionally produces a triangular area of paravertebral dullness on the side of the thorax opposite the effusion, constituting Grocco's sign of this disease.

Clinical and autopsy studies have demonstrated that certain portions of the mediastinum suffer displacement more readily than do others. In this respect, the middle mediastinum, containing the pericardium and heart, is more mobile than is the posterior mediastinum, in which the aorta and its intercostal branches tend to anchor this portion of the mediastinum more securely in its normal position. Extensive shifting of the mediastinum is attended by lateral displacement of the trachea, which may be palpated in the episternal notch.

The *thoracic viscera* comprise the air passages, represented by the larynx, trachea, and bronchi; the essential organs of respiration, the lungs; and the circulatory organs, represented by the pericardium and heart with the great vessels arising from its base.

The *larynx* must be considered in conjunction with the trachea and bronchi, as it constitutes an essential portion of the air passages. This important structure occupies a position in the neck between the base of the tongue and the trachea, and opposite the bodies of the fourth, fifth, and sixth cervical vertebræ. In the ventral portion of the neck the larynx forms a visible prominence, the pomum Adami, lateral displacement of which is often significant of intrathoracic disease. Similarly, abnormalities in the respiratory movements of the larynx are often the deciding factor in the determination of the cause of dyspnea; while a palpable tug of the larynx is not infrequently symptomatic of aortic aneurysm.

The cavity of the larynx contains the true and false vocal cords, intervening between which is the ventricle. The slight interval between the true cords constitutes the chink of the glottis or rima glottidis, variations in the dimensions of which influence the intensity of physical signs which are dependent upon the spoken voice for their production.

The mucous lining of the upper portion of the larynx is quite thick and vascular, and, in the presence of inflammatory affections of neighboring structures, is apt to swell from effusion into the submucous coat with the production of edema of the glottis, and stridulous respiration. The close adhesion of the mucous membrane to the true vocal cords prevents the effusion from extending below these structures.

The *trachea,* the second portion of the respiratory passage, has the form of a cylindrical tube with slight flattening of the posterior wall. It extends from its junction with the cricoid cartilage of the larynx opposite the sixth cervical vertebra to its bifurcation to form two primary bronchi at the level of the fourth dorsal vertebra. In the neck it is covered by the flat infrahyoid muscles, the isthmus of the thyroid gland, and the integument; while within the thorax it is covered by the thymus gland in the child, and by the remnant of this gland and by the arch of the aorta in the adult subject. The trachea of the adult is from four to four and one-half inches in length.

The trachea is a composite structure formed by from sixteen to twenty incomplete cartilaginous rings, invested externally by a fibrous membrane and lined internally by a mucous membrane containing numerous mucous glands, whose secretion prevents excessive dryness of the sensitive mucous lining of the tube. In the presence of tracheobronchial irritation and inflammation, these glands pour out an excessive, tenaceous secretion.

The free ends of the cartilaginous rings, of which the trachea is largely composed, are directed posteriorly, in which situation the deficiency in the wall of the tube is closed by transverse bundles of involuntary muscle, constituting the trachealis muscle of Todd and Bowman. The transverse bands of this muscle, the contraction of which serve to diminish the lumen of the trachea, are invested externally by bands of nonstriated muscular fibers which extend the entire length of the tube.

The *bronchi* pass obliquely downward and outward from their origin at the tracheal bifurcation to enter the roots of their respective lungs.

The *right bronchus,* approximately one inch in length, passes almost vertically downward to enter the root of the right lung opposite the fifth thoracic vertebra. This bronchus gives off an eparterial branch, situated just above the level of the pulmonary artery, to supply the upper lobe of the lung. The remaining branches of the tube are hyparterial, springing from the primary bronchus below the level of this artery. The vena azygos major courses upward behind the right bronchus; and, arching over the tube, joins the superior vena cava, which is in relation with the bronchus anteriorly. The right pulmonary artery occupies initially a position below the corresponding bronchus, and in its further course occupies a position anterior to it. As a result of its more vertical course, foreign bodies which are aspirated into the trachea have a tendency to enter the right bronchus.

The *left bronchus,* which is approximately two inches in length, enters the root of the left lung at a point opposite the sixth thoracic vertebra. In its course the left bronchus crosses the esophagus, thoracic duct, and descending aorta. The left pulmonary artery occupies a position at first superior to, and in its course finally comes to occupy a position posterior to, the bronchus. The arch of the aorta crosses above the left bronchus; and, in the presence of aneurysm of this portion of the vessel, is apt to produce a downward displacement of the bronchus which is responsible for the systolic tracheal tug which not infrequently characterizes this condition.

All of the branches of the left bronchus are hyparterial, whence the tube may be looked upon as equivalent to the portion of the right bronchus which lies distal to the eparterial branch. The first branch of the primary bronchus is distributed to the upper lobe of the lung, while the main stem of the tube supplies the lower lobe.

As the left bronchus possesses no eparterial branch, and as it is smaller and more deeply situated in the thoracic cavity than is the corresponding bronchus of the right side, and, moreover, as it forms a more acute angle with the trachea than does the right bronchus, physical signs arising within it are not conducted to the surface of the thorax with the same degree of intensity as are similar sounds generated within the right bronchus.

The *tracheobronchial lymph* nodes are collected into three principal groups along the course of the trachea and bronchi. A group of these glands occupies the angle between the trachea and right bronchus upon the right side, while a similar group occupies the angle between the trachea and the left bronchus upon the opposite side. A large group of glands is clustered in the angle which is formed by the tracheal bifurcation. Finally, the interbronchial nodes occupy the angles of bifurcation of the larger bronchi throughout the pulmonary parenchyma. With advancing age these glands become deeply pigmented as a result of the deposition of carbonaceous material from the pulmonary alveoli and finer bronchioles; and in the presence of malignant disease they become enlarged with the production of pressure signs.

The *lungs,* anchored by their respective roots, and covered upon their surfaces by the visceral pleura, as a result of their inherent elasticity and the negative pressure obtaining in the pleural cavities, completely fill these spaces during inspiration. Each lung possesses an apex, which ascends one to one and one-half inches above the clavicle beneath the dome of the pleura; a base which rests upon the convex superior surface of the diaphragm; a convex or costal surface, which is in contact with the anterior, lateral, and posterior thoracic walls; and an irregular internal surface, which bears the imprint of a number of structures contained within the mediastinum with which it is in relation.

The *left lung* is divided into two lobes by a deep fissure, which extends well in toward the root of the lung, and which is lined by a reflection of the visceral pleura. In inflammation of the pleura the portion of the membrane which dips into the fissure may be the only portion of the membrane involved, with the consequent production of a condition of interlobar pleurisy very difficult of detection by physical means.

The upper lobe of the left lung comprises a large portion of the external surface and the entire anterior border of the lung, while the lower lobe comprises the entire base and the greater portion of the posterior border of the lung. This is an anatomic fact of considerable importance, as during a physical examination it is frequently desirable to ascertain whether a morbid process having its inception in the apex or upper lobe of the lung has progressed to the lower lobe.

The *right lung* is divided into three lobes by two fissures. The upper lobe comprises the apex, a little more than half of the external surface, and the portion of the anterior border of the lung above the level of the fourth costal cartilage. The lower lobe comprises the entire base of the lung and the greater portion of the posterior surface. The middle lobe of the right lung is a wedge-shaped portion interposed between the upper and lower lobes, comprising the anterior portion of the external surface of the lung below the level of the fourth costal cartilage.

The external surface of each lung is convex; whereas the internal surface, which is in contact with the mediastinum, presents depressions corresponding to the mediastinal structures with which it is in relation.

The internal surface of each lung is marked by a rather deep depression, which receives the pericardium and heart. This depression is much more distinctly marked upon the left lung, owing to the projection of the heart to the left side of the median line of the thoracic cavity. Situated immediately above and behind the cardiac depression, each lung presents the hilus or pulmonary root, for the entrance of the primary bronchus with the accompanying vessels, lymphatics, and nerves of the lung; while extending downward from the hilus is a fold of the reflected pleura, the ligamentum latum pulmonis.

In addition to these impressions, the internal surface of the left lung is traversed by a fairly deep groove, which curves above the left bronchus and descends behind this tube, and which lodges the aorta. A second groove, which lodges the subclavian artery, passes upward from the aortic groove at the point where the latter arches over the pulmonary root.

The internal surface of the right lung is traversed by a groove which, arching over the right bronchus, lodges the vena azygos major. Extending upward from the fore part of this groove is a second groove which is traversed by the superior vena cava.

The lungs present certain individual differences which have an influence upon physical signs emanating from the two sides of the thorax. The apex of the right lung mounts to a greater height in the root of the neck beneath the dome of the pleura than does that of the left lung. Similarly, the base of the right lung occupies a higher level than does the base of the left lung. But, in addition to its slightly higher position with reference to the opposite lung, the right lung has a greater transverse diameter than its fellow of the opposite side. Moreover, the anterior border of the right lung is approximately vertical, approaching the median line; whereas the corresponding border of the left lung falls away from the median line in an oblique direction, exposing a portion of the right ventricle and pericardium in the interval which is created in this manner.

The extent to which the anterior borders of the lungs approach each other varies with the depth of inspiration and the integrity of the pulmonary parenchyma, as well as with the state of the mediastinal structures. In the absence of mediastinal disease, upon forced inspiration the anterior pulmonary borders come forward and cover the base of the heart and the great vessels arising therefrom and enveloped by the pericardium. At the completion of inspiration the anterior borders of the lungs are in contact from the lower border of the manubrium sterni to a point corresponding to the level of the fourth costal cartilage. At this stage of the respiratory cycle the anterior border of the right lung is vertical and parallel with the median line of the thorax, while the obliquely directed anterior border of the left lung covers all of the pericardium and heart save a limited portion corresponding to the lower third of the right ventricle. During tranquil respiration the anterior borders of the lungs do not come into contact at any point. Owing to the projection of the heart toward the left of the median line, the anterior border of the right lung is the more mobile of the two, and during tranquil inspiration approaches nearly to the median line behind the sternum.

Various factors may, however, modify the excursions of the anterior pulmonary borders. In the presence of excessive cardiac hypertrophy or extensive pericardial effusion the borders do not meet over any portion of the pericardium. The same condition obtains in the absence of cardiac or pericardial disease when the heart is pushed forward by disease in the posterior mediastinum. Moreover, the excursion of the pulmonary borders is restricted by the formation of adhesions between visceral and parietal pleura, or by cirrhotic changes in the pulmonary parenchyma.

As the anterior border of the right lung enjoys a greater freedom of movement than does the corresponding border of its fellow, so also the lower border of the right lung expands more freely than does that of the left lung. The apex of the left lung, on the contrary, expands more freely than does that of the right lung. During both tranquil and forced respiration the posterior portions of the lungs remain quietly in relation with the walls of the pleural cavities, while the apices, anterior borders, and bases expand and recede with inspiration and expiration. The excursion in any of these directions is apt to become deficient as the result of traction from adhesions or retrogressive changes in the lung.

During tranquil respiration a volume of air varying from 400 c.c. to 500 c.c. enters and leaves the lungs, constituting the *tidal air*. With the deepest possible inspiration 1,500 c.c. of additional air can be accommodated in the lungs, the *complemental air;* and during forced expiration 1,500 c.c. can be expelled above the tidal air. Following complete expiration there remains in the normal lungs from 1,000 c.c. to 1,200 c.c. of air which cannot be exhaled, constituting the *residual air.* During ordinary tranquil respiration at the end of expiration the lungs contain approximately 2,500 c.c. of air, the *stationary air.*

The *vital capacity* of the lungs, representing the maximum volume of air which can be expelled by forced expiration following full inspiration, in the normal subject amounts to from 3,500 c.c. to 4,000 c.c. In the presence of chronic pulmonary disease, and especially in cases of imperfectly compensated cardiac disease, the vital capacity is reduced to a variable degree.

The internal structure of the lung is very intimately related to many symptoms and signs which are manifested during disease of these organs. The primary bronchus, which enters the lung at the hilus, divides dichotomously until, after repeated divisions, very fine branches termed bronchioles are formed. The bronchioles have no communicating branches with the result that when an obstruction of one branch develops, air is withheld from the vesicles in which it terminates, and a condition of localized atelectasis inevitably results.

In the bronchiole there is a gradual transition of the stratified columnar epithelium of the bronchus into simple columnar epithelium, which in turn, near the distal end of the terminal bronchiole, gives place to small groups or islands of flat epithelial cells, *respiratory epithelium.* The epithelial lining of the terminal bronchiole is supported by a thin basement membrane, beneath which is a tunic containing numerous elastic fibers and circularly disposed bands of involuntary muscular fibers, spasm of which possibly plays a part in the production of the paroxysm of bronchial asthma.

Each terminal bronchiole terminates in an irregularly pyramidal chamber, the *infundibulum,* which constitutes the vascular unit of the lung. The walls of the infundibula are studded with a series of minute pouches, the *alveoli,* which are lined with a single layer of flat respiratory epithelium, supported by a delicate basement membrane containing numerous elastic fibers. These elastic fibers render the infundibular walls very resilient, and play an important part in the expansion and recession of the lung during respiration.

Each infundibulum is invested by a dense capillary plexus derived from branches of the pulmonary artery, which accompany the bronchi and bronchioles in their ramifications throughout the lung. The capillary plexus surrounding each infundibulum is disposed in a single layer, and there is no communication with the vessels of adjacent infundibula. The blood content of these capillaries is very intimately exposed to the air in the infundibula, as they are separated only by three very thin membranes; namely, the endothelium of the capillary wall, the delicate basement membrane of the infundibulum, and the single layer of infundibular epithelium. In hypertrophic emphysema, when the interalveolar septa are destroyed, as the capillary plexus of each infundibulum is distinct and has no communication with those of adjacent infundibula, the amount of blood exposed to the air in the large cavities which are formed by coalescence of several infundibula is considerably reduced, leading to dyspnea upon slight exertion in this class of patients.

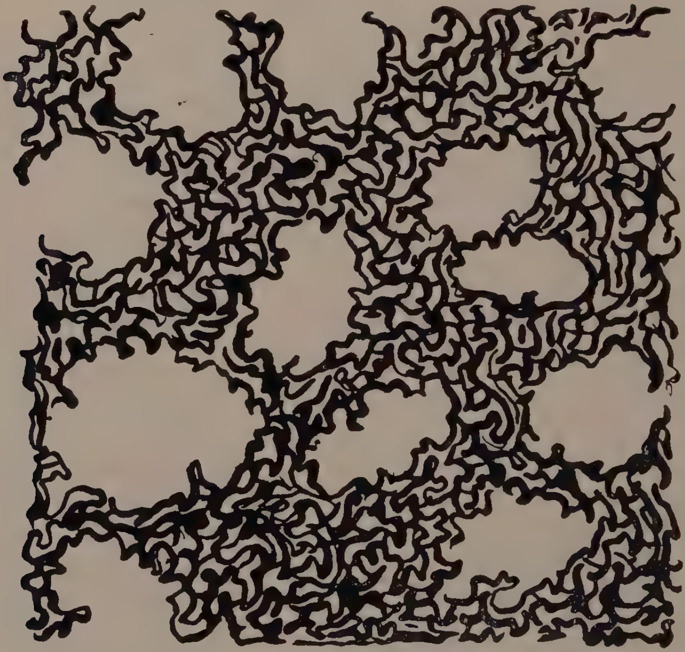


Fig. 3.—Pulmonary capillaries. (Brown, after Böhm, Davidoff, and Huber.)

The *lymphatics* of the lungs drain into the tracheo-bronchial lymph nodes and the mediastinal glands, with the result that these glands are early involved in tuberculous infection of the lungs, or when the lung is the seat of malignant disease. Similarly, these glands serve as filters for the irritant dusts which are conveyed to them by phagocytes from the finer bronchioles and infundibula in pneumonokoniosis.

While the major portion of blood traversing the lungs is contained in the various branches of the pulmonary arteries and veins, these vessels are concerned only to a negligible extent in the nutrient supply of the pulmonary parenchyma. The nutrient arteries of the lung are the bronchials, which, after traversing the hilus of the lung, follow the course of the bronchi, supplying these tubes and the surrounding interlobular tissues, as well as the bronchial lymph nodes. The venous return from these vessels is conveyed to the hilus by the bronchial veins, which in turn empty into the right and left azygos veins.

The *nerve supply* of the lungs is derived from the pneumogastrics and the sympathetic system, the function of respiration being presided over by the respiratory center in the medulla oblongata. In addition, the phrenic nerves supplying the diaphragm and the intercostal nerves supplying the muscles concerned in elevation of the ribs are coordinated with this center.

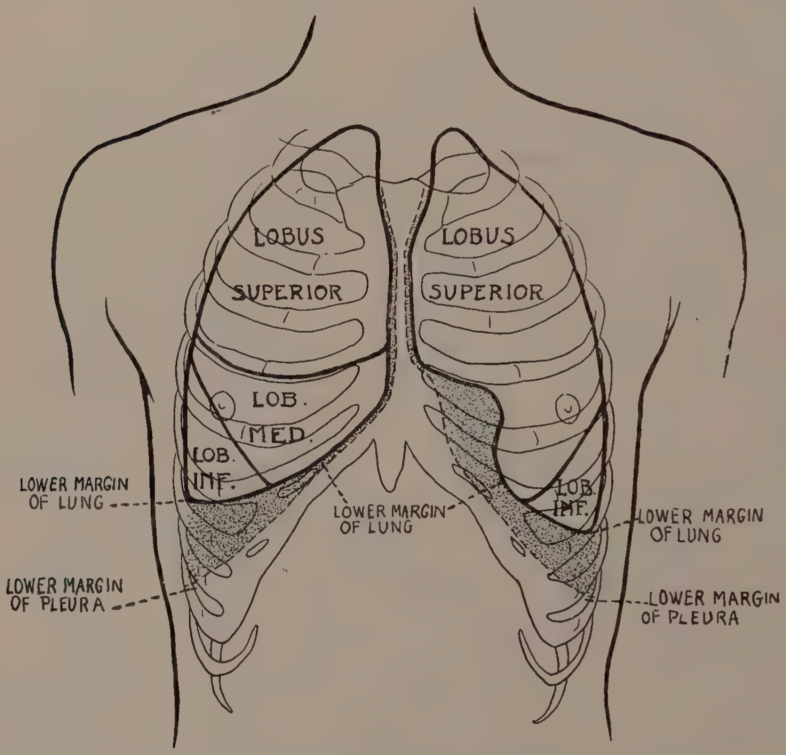
TOPOGRAPHICAL ANATOMY OF THE THORAX

The Pleura.—The surface markings of the pleura correspond to a line drawn from either sternoclavicular articulation downward and inward to the transverse ridge which marks the junction of the manubrium and gladiolus of the sternum. Thence the anterior borders of the reflections of the pleural membranes pass vertically downward slightly to the right of the median line to the level of the fifth intercostal space. At this point upon the surface of the thorax the two membranes separate, and in their further course diverge the one from the other.

The right pleura continues vertically downward almost to the junction of the gladiolus and the ensiform cartilage, whence it pursues an oblique course, passing outward, downward, and backward toward the vertebral column. In its course the lower border of the pleura crosses the seventh rib in the midclavicular line, the ninth rib in the midaxillary line, and the eleventh rib in the scapular line.

At the level of the fourth intercostal space the left pleura pursues a course toward the left and then downward to gain the posterior surface of the sixth costal cartilage in the left parasternal line. The membrane crosses the sixth costal cartilage vertically and from its lower border is reflected downward, outward, and backward toward the vertebral column, occupying a slightly lower level than does the lower border of the right pleura. In the interval which remains between the left sternal border and the left pleural membrane in the fifth intercostal space, an interval which corresponds accurately with the *incisura cardiaca* of the anterior border of the left lung, a limited portion of the right ventricle is brought into direct relation with the anterior thoracic wall.

The superior limit of the supraclavicular portion of the pleura, representing the dome of the pleural cavity, is indicated by a line drawn obliquely upward and outward from the sternoclavicular articulation upon either side, crossing the lower portion of the root of the neck so as to curve upward and descend to the spine of the seventh cervical vertebra. The maximum height of the curve, which corresponds to the apex or dome of the pleural cavity, is one to one and one-half inches above the clavicle.


LOBUS
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LOWER MARGIN OF LUNG
LOWER MARGIN OF PLEURA
LOWER MARGIN OF PLEURA

Fig. 4.—Illustrating the normal borders of the lungs and the location of the interlobar fissures. Anterior view. (Pottenger, after Corning.)

Upon the posterior surface of the thorax the course of the pleural reflection is represented by a vertical line drawn along either side of the vertebral column from the level of the seventh cervical vertebra to the articulation of the eleventh rib with the vertebral column, whence the line is continued downward and outward in a gentle curve to meet the line of reflection of the anterior portion of the membrane.

The Lungs.—The borders of the lungs correspond accurately with the line of reflection of the pleura, save that inferiorly the lower borders of the lungs fall short of the pleura by one intercostal space, being found at the level of the sixth rib in the midclavicular line, the eighth rib in the midaxillary line, and the tenth rib in the scapular line. The interval between the lower border of the lung and the inferior limit of the pleural reflection upon either side, representing one intercostal space upon the surface of the thorax, is the *complementary sinus,* into which the lower border of the lung descends during forced inspiration. These surface

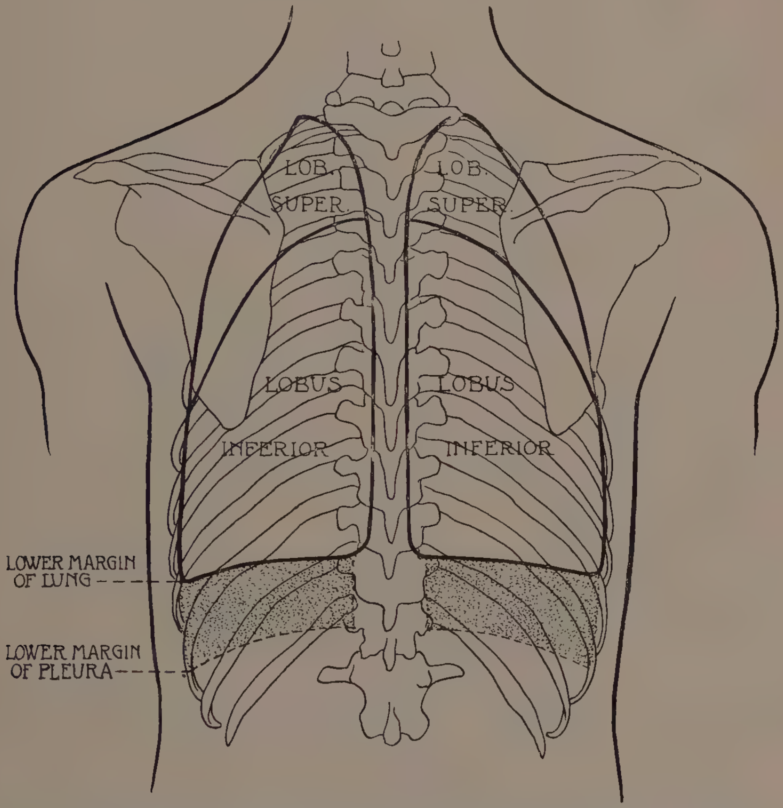


Fig. 5.—Illustrating normal borders of the lungs and interlobar fissures. Posterior view. (Pottenger, after Corning.)

markings should be borne in mind when determining the total expansion of the lungs by percussion.

Fissures and Lobes of the Lungs.—The position of the great fissure, which is common to both lungs and which intervenes between the upper and lower lobes, is represented by a line drawn upon the surface of the thorax from the spinous process of the third thoracic vertebra obliquely downward and forward to the lower border of the sixth rib in the midclavicular line. The shorter fissure, intervening between the upper and middle lobes of the right lung is represented by a line drawn upon the surface of the thorax from the posterior axillary line in the fourth intercostal space almost horizontally forward to meet the sternum at the level of the fourth right costal cartilage.

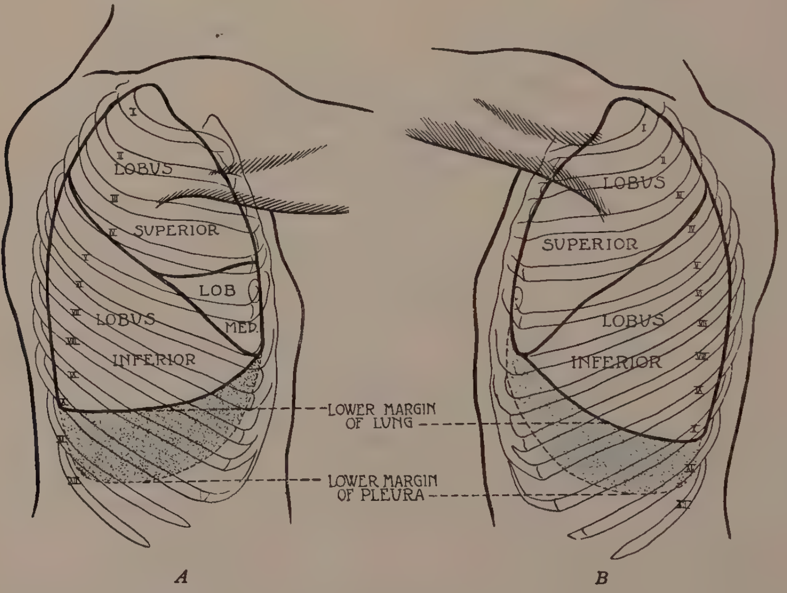


Fig. 6.—Illustrating the normal borders of the lungs and the location of the interlobar fissures. Lateral view. *A*, right; *B*, left. (Pottenger, after Corning.)

Trachea and Bronchi.—The course of the trachea corresponds to a broad line passing vertically downward from the upper margin of the manubrium sterni to the level of the upper border of the second rib in the median line of the thorax. At this point upon the surface of the thorax the trachea divides to form the two primary bronchi, which diverge from each other in a downward and outward direction, the right bronchus inclining more directly downward than does the left.

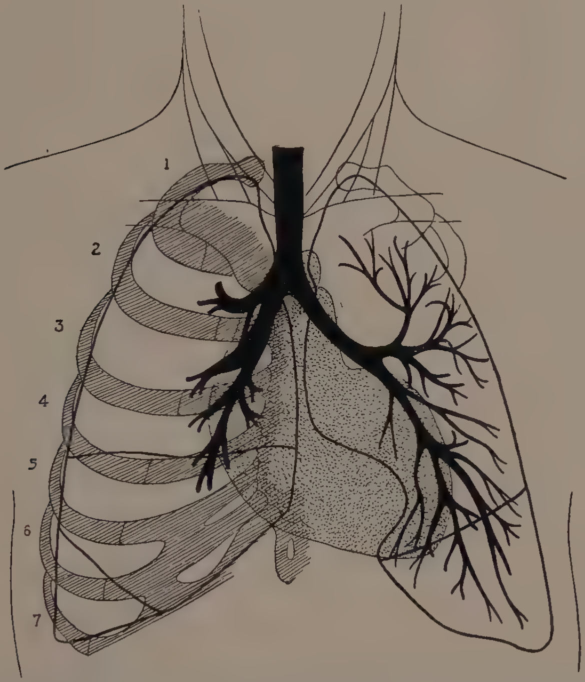


Fig. 7.—Showing the position of the bifurcation of the trachea and the peri-tracheal and peri-bronchial glands projected upon the anterior surface of the chest in a young adult. (Pottenger, after Gerhartz.)

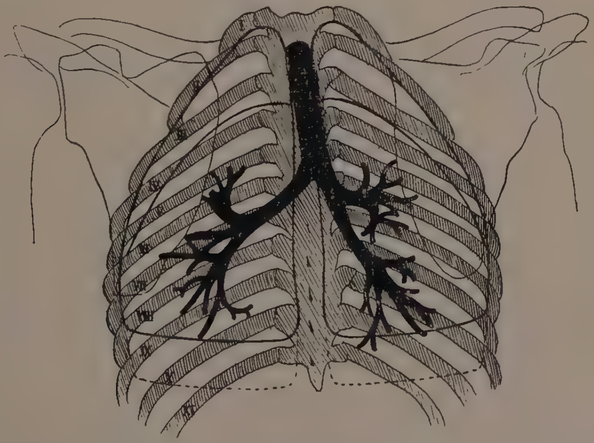


Fig. 8.—Showing the position of the bifurcation of the trachea with the peri-tracheal and peri-bronchial glands projected upon the posterior surface of the chest in a young adult. (Pottenger, after Piersol.)

ANATOMIC LANDMARKS OF THE THORAX

The thorax presents a number of anatomic structures which are visible or palpable, and which may be utilized as landmarks in the localization and description of morbid conditions arising within the thoracic cavity.

At the superior border of the manubrium sterni there is normally a visible depression of moderate depth, occupying the interval between the sternal attachments of the sternomastoid muscles, the *episternal* or *suprasternal notch*, which is occasionally the site of abnormal pulsations. At the completion of expiration the superior border of the manubrium, which limits the episternal notch inferiorly, occupies a position on a level with the disk between the second and third thoracic vertebræ. The interval between the vertebral column and the notch, representing the inlet of the thorax, measures approximately two inches in the normal subject.

At the inferior extremity of the ensiform cartilage there is a second visible depression, the *scrobiculus cordis,* or pit of the stomach, which corresponds to the level of the midpoint of the body of the ninth thoracic vertebra.

The *sternum* occupies the median line of the anterior surface of the thorax, surmounted by the episternal notch, and with the scrobiculus cordis at its lower extremity. Of variable prominence in different subjects, and readily palpable throughout its entire extent, the sternum is approximately six inches in length in the normal adult.

The *angulus Ludovici,* or *angle of Louis,* is a transverse ridge upon the sternum, which marks the junction of the manubrium and the gladiolus. It is usually visible upon the surface of the thorax and is always palpable. This ridge corresponds to the level of the junction of the second costal cartilage with the sternum, and it is frequently utilized as a starting point in counting the ribs and intercostal spaces.

The *clavicle,* at all times a conspicuous landmark upon the anterior chest wall, in apical lesions of the lungs becomes very prominent, contrasting markedly with the *supraclavicular fossa* above it and with the *infraclavicular fossa* situated immediately below the bone.

Upon either side of the thorax there are twelve *ribs* and eleven *intercostal spaces.* The first rib, situated rather deeply beneath the clavicle, is palpated with difficulty; but the remaining ribs are readily palpable and commonly visible in the form of moderate protrusions in the lower axillary and infraaxillary regions. Each intercostal space is named in accordance with the number of the rib immediately above it; hence the first intercostal space occupies the interval between the first and second ribs.

In *counting the ribs* one of several methods may be employed. It is convenient in counting the ribs upon the anterior surface of the thorax to commence the enumeration at the angle of Louis, which corresponds accurately to the second chondrosternal articulation. In counting the ribs upon the posterior surface of the thorax the inferior angle of the scapula may be utilized as a point of departure, as the tip of this bone overlies the seventh rib when the thorax is in repose. In the enumeration of the ribs upon the lateral aspect of the thorax the highest digitation of the serratus magnus muscle becomes the normal landmark, as this portion of the muscle overlies the sixth rib. The muscle may be rendered tense by abduction of the arm to a horizontal position. Certain clinicians prefer to proceed from below in counting the ribs, beginning with the extremity of the twelfth rib, which is palpable in the majority of subjects.

The *mammary gland* in the male subject is a rudimentary structure. In the female subject, however, it is well developed, and extends from the third to the seventh intercostal space in the midclavicular line. In the male subject the *nipple* is a reliable guide to the fourth intercostal space; but in the female, owing to the pendulous condition of the breast, it is an unreliable landmark.

The *scapula* overlies the dorsal aspect of the bony thorax, extending along the vertebral column from the second to the seventh rib. Always readily palpable, the bone stands out prominently in the form of the ''winged scapula'' of the alar thorax of chronic phthisis.

The *vertebral column,* or *spine,* in persons of excessive muscular development is represented by a linear, median furrow, and the spinous processes are palpated with difficulty. But in thin subjects and in children many of the spinous processes are visible with the patient in the erect posture; and upon bending the trunk forward they are readily palpated and counted. The spinous process of the seventh cervical vertebra is always a conspicuous landmark, and it may be employed as a point of departure in the enumeration of the vertebræ. Also the inferior angle of the scapula, which corresponds to the level of the spinous process of the seventh thoracic vertebra, may be utilized as a starting point in the enumeration. Lateral curvature of the vertebral column (scoliosis) or abnormal degrees of anteroposterior curvature (lordosis) may be encountered in connection with the thoracic deformities of rickets, hypertrophic emphysema, and in other forms of pulmonary disease.

TOPOGRAPHICAL LINES AND REGIONS

For purposes of clinical description and for convenience in localizing lesions arising within the thorax, a number of regions may be outlined upon the surface of the thorax by means of arbitrary vertical and horizontal lines. It is of the first importance that the student should become thoroughly familiar with the relations of the thoracic viscera to these several regions.

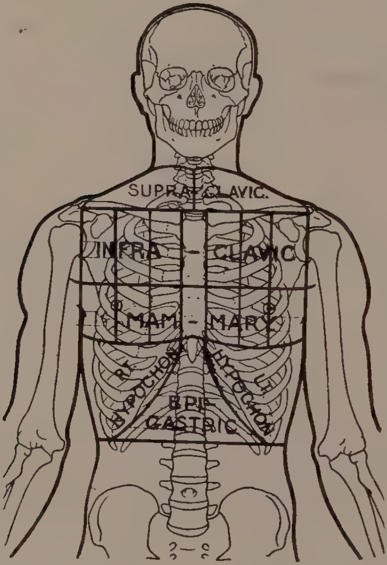


Fig. 9.—Topographic regions of the thorax. (Anterior view.)

Vertical Lines.—The *midsternal line* is a vertical line erected upon the anterior aspect of the thorax, traversing the midportion of the sternum from the midpoint of the superior border of the manubrium sterni to the tip of the ensiform cartilage.

The *sternal line* conforms to the lateral border of the sternum from the sternoclavicular articulation to the junction of the gladiolus with the ensiform cartilage, whence it is continued in a direction downward and outward along the course of the lower border of the costal arch to meet the anterior axillary line.

The *midclavicular line* is a vertical line dropped upon the anterior thoracic surface from the midpoint of the clavicle. It frequently, though not invariably, passes through the nipple and hence is frequently termed the *nipple line*, or the *mammary line*.

The *parasternal line* is a vertical line occupying a position upon the anterior aspect of the thorax midway between the sternal and the midclavicular lines.

The *anterior axillary line* is a vertical line dropped along the lateral thoracic wall from the anterior fold of the axilla.

The *midaxillary line* is a vertical line which passes down the lateral wall of the thorax from the apex of the axilla.

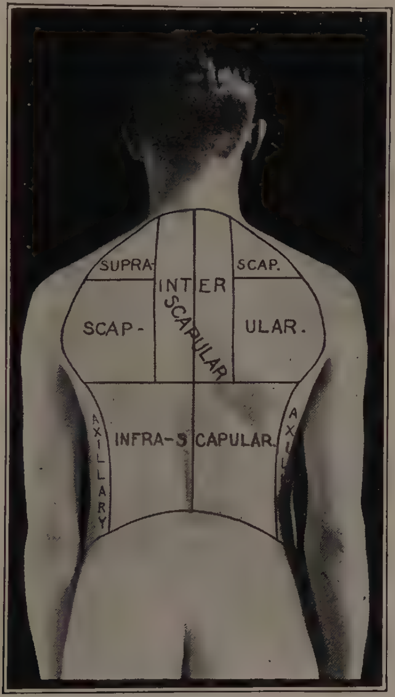


Fig. 10.—Topographic regions of the thorax. (Posterior view.)

The *posterior axillary line* is a line which is dropped along the lateral thoracic wall from the posterior fold of the axilla.

The *scapular line* is a vertical line drawn upon the posterior surface of the thorax in such fashion as to pass through the tip of the inferior angle of the scapula.

The *midspinal line* is a vertical line which conforms to the series of spinous processes of the vertebræ.

Horizontal Lines.—The *cricoclavicular line* is drawn from the prominence of the cricoid cartilage outward and with a slight inclination downward to meet the prominence caused by the outer extremity of the clavicle.

The *clavicular line* is a horizontal line passing outward from the sternoclavicular articulation, conforming to the course of the clavicle.

The *third costal line* is drawn horizontally outward from the sternal line at the level of the third costal cartilage to meet the anterior axillary line.

The *sixth costal line* is a horizontal line commencing at the sternal line at the level of the sixth chondrosternal articulation, which, after crossing the anterior axillary and midaxillary lines, terminates at the posterior axillary line.

The *scapular spinal line* is a horizontal line projected outward upon the posterior thoracic wall from the scapular line, and conforming to the course of the spine of the scapula.

The *infrascapular line* is a line drawn horizontally across the posterior surface of the thorax at the level of the inferior angles of the scapulæ.

The *twelfth dorsal line* is drawn from the point overlying the spinous process of the last thoracic vertebra, passing downward and outward to meet the posterior axillary line.

Regions of the Thorax.—Through the medium of these various vertical and horizontal lines, a number of regions are mapped out upon the surface of the thorax.

The *sternal region* overlies the sternum and is bounded above by the episternal notch, below by the scrobiculus cordis, and laterally by the sternal lines. The upper portion of the sternal region, overlying the manubrium sterni, contains the anterior borders of the lungs, together with the bifurcation of the trachea, and the arch of the aorta. This region also is traversed by the left innominate vein and the beginning of the superior vena cava. The lower portion of the sternal region, corresponding to the distribution of the gladiolus, overlies the greater portion of the right ventricle together with the edge of the left ventricle, as well as a portion of the anterior borders of both lungs. Within this region also are found the origin of the pulmonary artery and the ascending aorta, and the space also includes the right auricle. This region overlies the attachment of the pericardium to the diaphragm, and also a limited portion of the left lobe of the liver.

The *supraclavicular region* lies in the base of the neck, above the clavicle, bounded above by the cricoclavicular line and inferiorly by the clavicular line. These regions, upon either side, contain the apices of the lungs, the common carotid and subclavian arteries, together with the subclavian and jugular veins.

The *infraclavicular region* is situated immediately below the clavicle. It is bounded above by the clavicular line, below by the third costal line, internally by the sternal line, and externally by the anterior axillary line. The anatomic structures lying within the limits of the infraclavicular regions differ upon the two sides of the thorax. The *left infraclavicular region* overlies the upper lobe of the left lung; and, adjacent to the left sternal border, a portion of the left auricle and the left pulmonary artery, as well as a portion of the left bronchus. The *right infraclavicular region* contains within its limits the greater portion of the upper lobe of the right lung, with the termination of the right bronchus, and the right pulmonary artery.

The *mammary region* lies immediately below the infraclavicular region, upon either side of the thorax, limited above by the third costal line, below by the sixth costal line, internally by the sternal line, and externally by the anterior axillary line.

As in the case of the infraclavicular regions, so also in the mammary regions the structures which are contained within the limits of these regions vary widely upon the two sides of the thorax. The *left mammary region* contains a portion of both lobes of the left lung, as well as the greater portion of the heart and pericardium, the latter partially overlapped by the anterior border of the left lung. The *right mammary region* contains a portion of the right auricle and right ventricle, partially overlapped by the anterior border of the right lung, which occupies the major portion of this region. The right lobe of the liver, clothed by the diaphragm, ascends in this region as high as the upper border of the fifth rib in the midclavicular line.

The *hypochondriac region* lies immediately below the mammary region, upon either side of the thorax, between the sixth costal line, the anterior axillary line, and the downward and outward continuation of the sternal line along the line of the costal arch. The contents of the regions vary upon the two sides of the thorax.

The *left hypochondriac region* contains the complementary sinus into which the lower border of the left lung descends during inspiration, the cardiac end of the stomach, the diaphragm, and the tip of the left lobe of the liver. The *right hypochondriac region*, in addition to the lower border of the right lung, contains within its limits the right lobe of the liver, covered by the diaphragm.

The *axillary region* occupies the superior portion of the lateral aspect of the thorax, limited superiorly by the apex of the axilla, inferiorly by the sixth costal line, anteriorly by the anterior axillary line, and posteriorly by the posterior axillary line. This region upon both sides of the thorax contains only pulmonary tissue, hence upon percussion it yields a frankly resonant note.

The *infraaxillary region,* lying immediately below the axillary region upon the lateral aspect of the thorax, is bounded by the anterior and posterior axillary lines, the sixth costal line, and the downward prolongation of the sternal line along the costal arch.

The *left infraaxillary region* contains the lower portion of the left lung, the lower border of which reaches the eighth rib in the midaxillary line. The space also contains the diaphragm, and below this muscle a portion of the stomach and the spleen. The *right infraaxillary region* contains, in addition to the right lung and the diaphragm, a portion of the right lobe of the liver.

The *suprascapular region* overlies the supraspinous fossa of the scapula upon the posterior wall of the thorax, and is limited below by the scapular spinal line. The only structure within this region is the pulmonary apex upon either side of the thorax.

The *scapular region,* overlying the infraspinous fossa of the scapula, is limited superiorly by the scapular spinal line and inferiorly by the infrascapular line. This region upon either side of the thorax contains the posterior voluminous portions of the lungs, including a portion of both upper and lower lobes.

The *infrascapular region* upon either side of the thorax is limited superiorly by the infrascapular line, inferiorly by the twelfth dorsal line, and externally by the posterior axillary line. The anatomic structures within the limits of this region vary upon the two sides of the thorax.

The *left infrascapular region* overlies the lower lobe of the left lung, its inferior margin reaching the tenth rib in the scapular line. This region also contains the thoracic aorta in the lower portion of its course, and below the diaphragm a portion of the left kidney and of the spleen. The *right infrascapular region* contains, in addition to the lower lobe of the right lung, a portion of the right lobe of the liver, and of the right kidney.

The *interscapular region* occupies the interval upon either side of the thorax between the scapular line and the midspinal line, limited inferiorly by the infrascapular line. The structures contained in the two regions are different upon the two sides of the thorax.

The *left interscapular region,* in addition to the left lung, contains a portion of the left bronchus; and, near the midspinal line, the descending thoracic aorta, the esophagus, and the thoracic duct. The *right interscapular region* overlies the right lung and right bronchus in a portion of its course. The trachea extends downward in front of the bodies of the thoracic vertebræ from its junction with the larynx opposite the sixth cervical vertebra to the fourth dorsal vertebra, where it bifurcates to form the bronchi. The tracheobronchial glands are clustered about the angles formed by the bifurcation.

CHAPTER II

CLINICAL ANATOMY AND PHYSIOLOGY OF THE CIRCULATORY SYSTEM

THE HEART

The heart, the great muscular pump by which the column of blood is propelled through the vessels, is a roughly conical organ, situated obliquely in the middle mediastinum between the lungs, whose anterior borders partially overlap its ventral surface. The heart presents for examination a base, which is directed upward and toward the right, an apex, which is directed downward and toward the left, and three borders; namely, right, left, and inferior.

In the absence of disease, the position of the heart is influenced largely by the physical make-up of the individual. In the subject of normal habitus the heart occupies a moderately oblique position in the middle mediastinum, approximately one-third of the organ lying to the right of the median line, and two-thirds to the left of this line. In the subject of hypersthenic habitus, characterized by a short, deep thorax with an obtuse costal angle, the heart occupies a more transverse position in the thorax. In such cases the inferior border of the organ seems to be sunk into the dome of the diaphragm to a variable degree, and the heart may extend well to the left of the median line of the body. In persons of the asthenic habitus, again, the physical make-up which is characterized by a long, shallow thorax with acute costal angle, the heart occupies an almost vertical position, very nearly in the midline of the thorax.

The heart is a hollow muscular organ, comprising four chambers or cavities, an *auricle* and a *ventricle* upon either side, the cavities of the one side being separated from those of the opposite side by a muscular septum. Each auricle, on the contrary, communicates with the corresponding ventricle through the auriculoventricular orifice. The left auricle and ventricle contain arterial blood, while the right auricle and ventricle contain venous blood.

The external surface of the heart presents definite markings, in the form of transverse and longitudinal furrows, which accurately indicate the internal subdivisions of the organ. The auriculoventricular groove encircles the heart transversely at a point somewhat nearer the base than the apex of the heart. This groove corresponds upon the surface of the heart to the site of the auriculoventricular septa, and lodges the coronary arteries. The interventricular furrows pass downward from the auriculoventricular furrow in the direction of the long axis of the heart, upon its anterior and posterior surfaces, respectively, terminating at the inferior sharp margin of the heart, which they reach a little distance to the right of the apex.

The cardiac wall is formed by bundles of specialized involuntary muscular fibers, the cardiac muscle, or *myocardium,* the bundles of which have a characteristic arrangement. The muscular bundles take origin from the rings which guard the auriculoventricular and aortic orifices. In the auricles the bundles are arranged in two layers, a superficial and a deep lamina, the latter composed of looped and annular fibers.

In the ventricles the bundles are likewise disposed in superficial and deep strata. The deep bundles encircle the ventricles, while the superficial fibers pursue a spiral course, coiling inward at the apex in the form of a whorl or vortex.

The myocardium possesses certain anatomic and physiologic peculiarities which serve to differentiate it from skeletal muscle. Anatomically the muscle fibers are striated; they are separated by a minimal amount of fibrous connective tissue; and the bundles present numerous anastomoses. Physiologically the myocardium presents the inherent properties of *rhythmicity*, the power of producing the impulse to contraction; *irritability,* the ability to respond to an adequate stimulus, *contractility*, the power of altering its conformation in response to stimulation; and *tonicity,* or a state of partial contraction which is constant.

The highest point of rhythmicity of the heart is represented by the sinoauricular node, the normal pace-maker of the heart. Other portions of the myocardium are likewise rhythmic, the relative rhythmicity of its several parts being represented by the auriculoventricular node, auriculoventricular bundle, auricular musculature, and ventricular musculature.

The myocardium is not of uniform thickness. The auricular walls are thinner than are those of the ventricles, usually measuring 2 to 3 millimeters in thickness. Similarly, the ventricles are not of uniform thickness, the right ventricuar wall usually measuring 5 to 6 millimeters in thickness, whereas the left ventricular wall usually measures 10 to 15 millimeters (Letulle).

The cavities of the heart are lined by a serous membrane, the *endocardium,* which is reflected over the cusps of the cardiac valves. The external surface of the myocardium is clothed by a similar serous membrane, the *epicardium,* which constitutes a portion of the visceral layer of the pericardium.

The *right auricle* consists of two portions: a principal cavity, the *sinus venosus,* or *atrium;* and an anterior recess, the *auricular appendix.* The walls of the right auricle represent the thinnest portion of the myocardium, and they readily dilate when an increased load is thrown upon the right side of the heart by an obstacle to the circulation of the blood through the pulmonary circuit. The interauricular septum presents a depression upon the inner wall of the auricle, the *fossa ovalis,* corresponding to the foramen ovale of the fetus. In certain cases of congenital heart disease, and indeed in 14 per cent of adults, this foramen does not close completely, its patency often producing obscure physical signs. The capacity of the right auricle averages sixty cubic centimeters, slightly exceeding the capacity of the left auricle. The right auricle communicates with the right ventricle by the auriculo-ventricular orifice, which is guarded by the tricuspid valve. It receives venous blood from the large superior and inferior venæ cavæ, the former returning blood from the head, neck, and upper extremities, and the latter returning blood to the heart from the trunk and lower extremities.

The *left auricle,* smaller than the right auricle, has thicker walls than the latter and hence its contractile power is greater. Like the right auricle, it is composed of an atrium and an auricular appendix. The left auricle communicates with the left ventricle by the bicuspid or mitral valve and it receives blood from the lungs through the large pulmonary veins. These large vessels usually empty their contents by four orifices in the base of the auricle; but not infrequently these orifices are reduced to three, the two left pulmonary veins terminating in a common trunk.

The *right ventricle,* triangular in contour, extends from the right auricle to the lower sharp border of the heart, reaching almost to the apex of the organ. Its anterior surface is convex and constitutes the major portion of the ventral aspect of the heart, the portion which is in relation to the anterior chest wall in the interval between the incisura cardiaca of the left lung and the left sternal border. The posterior surface of the ventricle is flattened and rests upon the central tendon of the diaphragm. The right ventricle has a capacity of about ninety cubic centimeters, and its walls are not as thick as are those of the left ventricle. The ventricular wall is thinnest in the region of the superior and internal angle of the ventricle, the *conus arteriosus,* which overlies the opening of the pulmonary artery. In addition to the opening of the pulmonary artery, which is guarded by the semilunar valves, the ventricle communicates with the right auricle by the right auriculoventricular orifice, which is guarded by the tricuspid valve.

The internal surface of the ventricle presents numerous muscular columns, *columnæ carneæ,* projecting from the myocardium and invested with endocardium. Three, or it may prove to be four, of these muscular columns are very well developed and constitute the *papillary muscles,* which are connected with the margins of the cusps of the tricuspid valve by delicate tendinous cords, *chordæ tendineæ.*

The *left ventricle,* longer, thicker, and more conical in shape than the right ventricle, forms only a limited portion of the anterior surface, but the major portion of the posterior surface of the heart. The inferior extremity of the left ventricle alone forms the apex of the heart. The capacity of the left ventricle varies from eighty to one hundred cubic centimeters, and its walls are thicker than any other portion of the myocardium.

The interior of the left ventricle is similar anatomically to that of the right ventricle. The ventricular cavity communicates with the left auricle by the left auriculoventricular orifice, which is guarded by the bicuspid or mitral valve; and anteriorly and to the right of this valve it presents the aortic orifice, guarded by the semilunar valves. The right cusp of the mitral valve intervenes between the auriculoventricular and the aortic orifices; and, in the presence of aortic insufficiency, this cusp is apt to become the target of two streams of blood entering the left ventricle in opposite directions, the one from the left auricle and the other regurgitating from the aorta, and to be thrown into vibration with the production of a murmur, the murmur of Austin Flint, which will be described in a subsequent paragraph.

The volume of blood expelled by the left ventricle varies with the state of the myocardium, the rate of venous return, the rate of cardiac contraction, and the degree of peripheral resistance.

The output per unit of time is essentially dependent upon the integrity of the myocardium. While in the presence of a normal heart muscle the ventricular output is usually equal to the demands which may be made upon this chamber of the heart, in myocardial degeneration the ventricle is incapable of responding adequately to a normal or increased demand, with consequent diminution of the ventricular output.

With a normal myocardium, the left ventricular output is greater in the presence of increased venous return to the heart. Under these circumstances the ventricular cavity is rapidly filled during early ventricular diastole and auricular systole, and its ejection time is moderately prolonged.

Similarly, in the case of the normal heart, increase in the cardiac rate up to a certain point is attended by an increased ventricular discharge per unit of time. However, in pathologic cases tachycardia is frequently attended by a diminished output per unit of time, a change which seems to be compensatory under these conditions.

The influence of increase of peripheral resistance in the general circulation upon the ventricular output varies with the degree of resistance and with the state of the heart muscle. A sound myocardium responds to increased peripheral resistance with a prolonged systole and an increased output of blood. In the normal and pathologic heart, however, there is obviously a point beyond which the heart cannot respond adequately to an increased load.

The weight of the heart is not uniform in the two sexes. In the male subject the usual weight of the normal heart approximates 300 grams, whereas in the normal female subject the usual weight is approximately 280 grams.

The Cardiac Valves

The left auriculoventricular, *bicuspid,* or *mitral valve,* intervening between the left auricle and left ventricle, consists of a muscular ring supporting two semilunar segments, or cusps. The concavity of each of these cusps is directed toward the ventricle; and when, during ventricular systole their free margins are approximated, the auriculoventricular orifice is securely closed, and regurgitation of blood from the ventricle into the auricle is effectually prevented. Inversion of the valve segments during ventricular systole is prevented by traction upon their free borders by the papillary muscles through the medium of the chordæ tendineæ.

The right auriculoventricular, or *tricuspid valve,* comprises three segments or cusps, supported at their bases by a muscular ring. Their action is in all respects identical with that of the mitral valve, and their inversion during ventricular systole is similarly prevented by contractions of the papillary muscles with their chordæ tendineæ.

The *aortic valve* consists of a muscular ring supporting three semilunar cusps, with their concave surfaces directed toward the lumen of the aorta. This valve is devoid of chordæ tendineæ and papillary muscles; but each segment is reinforced by a thin cartilaginous plate, the corpus arantii. The valve is closed by the force of the blood column which is ejected into the aorta during the systole of the left ventricle. Hence, the closure of this valve is diastolic, whereas that of the mitral and the tricuspid valves is systolic in time. Opposite each segment of the aortic valve the wall of the aorta presents a small pouch or dilatation, the *sinus of Valsalva,* from two of which arise the coronary arteries, which nourish the myocardium.

The *pulmonary valve,* which intervenes between the right ventricle and the pulmonary artery, is similar anatomically to the aortic valve and its closure is effected in the same manner.

The function of the cardiac valves is to maintain a constant flow of blood in one direction. Owing to the delicacy of their structure, they become ready targets for infectious material which may gain access to the blood stream. As a result of such infection, structural changes, resulting in permanent deformity of the cusps, are apt to ensue, thus destroying the integrity of one or more of the valves. Such changes are inevitably followed by the sequence of events described under the effects of chronic valvular disease. Or, in the absence of infection and structural alteration in the valve segments, in the presence of malnutrition or excessive physical strain the muscular rings at the orifices may stretch, with the result that the normal segments can no longer close the abnormally large orifice. In certain instances congenital deformities of one or more of the cardiac valves are the cause of imbalance of the circulation.

The Cardiac Nerves

The heart is supplied from two systems of nerves; namely, the vagus nerves arising in the medulla oblongata, and sympathetic nerves arising from the superior segments of the thoracic cord and passing by way of the white rami communicantes to the stellate ganglion, the inferior cervical ganglion, and the myocardium.

These two systems exert an antagonistic action upon the cardiac functions, the vagus serving to diminish the rate of cardiac contraction and exerting a negative influence upon the several functions of the myocardium, while the sympathetic tends to increase the heart rate and to exert a positive influence upon the several attributes of the heart muscle.

Extensive experimental studies on animals have been carried out in the effort to determine the relation of the extracardiac nerve fibers to the fibers of the S-A node, the A-V node, and the A-V bundle. It seems to have been established that the fibers of the right vagus communicate largely with the cells of the S-A node, while the left vagus fibers terminate in the cells of the A-V node and bundle. Hence it follows that the right vagus is concerned principally with rhythm production in the S-A node, while the left vagus presides over variations in A-V conduction. In man pressure on the right vagus will often have more influence over rate than conduction, while pressure on the left vagus will often have more influence on conduction.

Similarly, the right sympathetic fibers exert their chief influence upon the S-A node, resulting in an increase in auricular and ventricular rates, without any change in the conduction time, whereas the left sympathetic fibers exert their influence chiefly upon the A-V node, resulting in reduction of the conduction time.

The Conduction System.—Whether one accepts the neurogenic or the myogenic theory of the heart-beat, it is established that the impulse to contraction is elaborated in a small club-shaped mass of specialized tissue, situated in the myocardium which forms the posterior portion of the auricle between the openings of the superior vena cava superiorly and the coronary sinus inferiorly, the *sinoauricular node,* or pace-maker of the heart. Histologically the node is composed of fusiform muscle fibers, which interlace and which are embedded in connective tissue. The node is not entirely of muscular origin, however, as it contains numerous nerve fibers, and a few ganglion cells (Oppenheimer).

From the sinoauricular node, the impulse to contraction is communicated to the auricular musculature, in which a wave progresses downward in a regular manner, to reach another specialized mass of tissue in the posterior and right border of the interauricular septum, the *auriculoventricular node.*

From the A-V node a narrow strand passes across the auriculoventricular junctional tissues, constituting the *bundle of His.* From the junctional tissues, the bundle enters the interventricular septum, where it divides into two primary branches which pursue a course beneath the endocardium of the right and left ventricles. After numerous subdivisions, the terminal branches of the bundle terminate in an extensive arborization upon the inner walls of the ventricles.

The auriculoventricular bundle of His is the medium of transmission of the impulse which results in systolic contraction from the auricles to the ventricles. Section of the bundle results in dissociation of auricular and ventricular action. Lesions of the myocardium involving the integrity of the bundle result in delayed conduction of the impulse, in partial blocking of the impulse, or in complete heart block. The interval of time which is required for the transmission of the impulse over the bundle ranges from 0.11 to 0.19 second under normal circumstances.

The Cardiac Cycle.—The cardiac cycle or the sequence of events constituting a contraction of the heart comprises auricular systole, ventricular systole, and a brief pause, during which the entire myocardium is in a state of repose. During the passive state which precedes auricular systole, the auricles receive blood from the veins, and a portion of this blood passes through the open auriculoventricular valves into the ventricles. However, as the auricles receive a greater quota of blood than immediately passes through the valves into the ventricles, they become passively distended toward the end of the period of repose, or diastole. At this instant a contraction wave is initiated at the S-A node, which, passing progressively downward through the auricles, forces the auricular contents into the ventricles.

A contraction of the muscular walls of the great veins exerts a valvular action upon their orifices during the period of auricular systole, preventing the reflex of blood from the auricles during their contraction. In the presence of auricular engorgement, however, this valve action is not effective against the backward flow of blood during auricular systole.

The force of the blood which is propelled into the ventricles by auricular systole is insufficient to open the semilunar valves, and is expended in distending the walls of the ventricles. The dilatation of the ventricles which ensues upon the the expulsion of the auricular contents is followed by the initiation of ventricular contraction, which corresponds to auricular diastole. The A-V valves, which have become opened as the blood flowed into the ventricles from the auricles, are firmly closed and their cusps are prevented from inversion by the traction of the papillary muscles and chordæ tendineæ. As the pressure rises in the ventricles there is a brief period in which these chambers are completely closed, the presphygmic period, during which the pressure is sufficiently high to close the A-V valves, but not sufficient to open the semilunar valves.

When the pressure within the ventricles exceeds that in the aorta and pulmonary artery, the semilunar valves are forced open and the ventricular content is expelled into these large arteries, during the sphygmic period of the cardiac cycle. The ventricles are more or less completely emptied during this period, which is maintained for a brief interval after the blood is expelled from these chambers. The sphygmic period is followed by an abrupt relaxation of the ventricles, during which intraventricular pressure rapidly falls and the semilunar valves are forced into apposition by the pressure in the aorta and the pulmonary artery. For a brief interval the ventricles are again closed cavities, but at the completion of ventricular relaxation the auriculoventricular valves open and blood enters the ventricles during the period of repose of the entire heart.

As the ventricles relax a moderate negative pressure is established, which is assumed to be of slight aid to the pulmonary circulation. The degree of this pressure is inconstant, but it averages 20 mm. of mercury in most cases.

In the normal adult subject the entire cardiac cycle lasts 0.8 second, of which 0.1 second is represented by auricular systole, 0.3 by ventricular systole, and 0.4 by the period of repose, representing diastole of all four chambers of the heart. During periods of tachycardia, the increase in the heart rate occurs largely at the expense of diastole.

During its contraction the heart moves forward upon its long axis in a direction from left to right, and the apex tilts upward and toward the right. At the same time the auricles become smaller, while the aorta and pulmonary artery are distended and elongated. With the inception of diastole the heart passes backward and toward the left. The auricles are now distended, while the aorta and the pulmonary artery are collapsed and shortened.

The Pericardium

The pericardium is a conical seromembranous sac situated between the lungs in the middle mediastinum, and enveloping the heart and the great vessels arising from its base. With its apex directed upward and its base moored to the central tendon of the diaphragm by areolar tissue, the pericardium lies behind the sternum and the costal cartilages of the third, fourth, fifth, sixth, and seventh ribs of the left side. Anteriorly the pericardium is separated from the chest wall in the greater portion of its extent by the anterior borders of the lungs; but a small portion of the sac is in direct relation with the thoracic wall in the interval between the left sternal border and the anterior border of the left lung in the fourth and fifth intercostal spaces. The posterior surface of the sac is in contact with the bronchi, the esophagus, and the thoracic aorta.

In the presence of inflammatory affections of the structures with which the pericardium is in contact, adhesions are apt to form, causing displacement of the sac with its contents. Or, adhesions may form between the sac and the chest wall, resulting in retraction of the surface of the thorax.

The pericardium is composed of an outer fibrous coat, which is prolonged upward upon the great vessels which arise within the sac, finally to blend with the deep cervical fascia; and of an internal serous coat, which is reflected on to the heart as the epicardium. This layer secretes a small amount of serous fluid, enabling the parietal and visceral layers of the pericardium to glide noiselessly over each other during the cardiac contractions. In the presence of inflammation of the membrane, however, the membranes become dry and covered with exudate, with the production of a to-and-fro friction sound, corresponding fairly closely with the cardiac systole and diastole.

In pericarditis with effusion the fluid is prone to collect on either side of the heart and below it. The pericardium and heart are separated by the diaphragm from the left lobe of the liver, and upon the extreme left, corresponding to the position of the apex of the heart, from the stomach. Hence in pericardial effusions the fluid gravitates toward the left, as the diaphragm yields more readily to pressure on this side than it does upon the right, where it is in relation to the left lobe of the liver.

The Coronary Arteries

The blood supply of the myocardium is derived from the coronary arteries, right and left, arising from the anterior and the left posterior sinus of Valsalva, respectively. After traversing the auriculoventricular groove, and supplying the auricle, the descending branch of the right coronary is given off at the posteroinferior interventricular furrow. This large branch descends in the interventricular groove to the apex of the heart, supplying branches to both ventricles and to the interventricular septum. Similarly, the descending branch of the left coronary artery descends in the anterosuperior interventricular furrow toward the apex of the heart, supplying similar branches to both ventricles.

The venous flow from these vessels is returned to the right auricle by the coronary sinus, a relatively large venous channel, which empties into the auricle at a point situated between the opening of the inferior vena cava and the right auriculoventricular orifice. A negligible venous return reaches the auricles and ventricles through the veins of Thebesius, minute venous channels, arising within the myocardium and discharging their contents into the cavities of the heart.

The maintenance of the coronary circulation depends upon the arterial pressure obtaining in the proximal aorta, the rate of contraction of the heart, the integrity of the coronary arteries, and the state of the root of the aorta whence the coronary arteries spring.

The flow through the coronary circuit is markedly influenced by variations in arterial pressure in the general circulation. With forceful ventricular contractions a large volume of blood is expelled into the aorta and intraaortic pressure is elevated, with consequent improvement in the coronary circulation. Likewise, during ventricular systole the vessels traversing the myocardium are compressed, and their content is driven onward in the direction of least resistance; namely, toward the venous return. Thereafter, upon diastole of the ventricle, the higher pressure obtaining in the aorta rapidly refills the coronary vessels.

In the presence of an intact myocardium, increased rate of cardiac contraction is usually attended by an acceleration of the coronary flow, as the increased output raises the pressure in the proximal aorta. However, in the tachycardia of cardiovascular disease, conditions are seldom ideal for the promotion of the coronary circulation.

In cases of endarteritis and sclerosis of the coronaries the myocardium does not receive an adequate circulation, predisposing to local or general retrogressive changes in the muscle. Similarly, in the absence of sclerosis of the coronary vessels, fibrosis of the proximal aorta may result in partial occlusion of the orifices of the coronary arteries, with a similar impairment of cardiac nutrition.

Local obstruction of the coronary circulation may arise from embolism or thrombosis involving these arteries, accidents which are attended by urgent symptoms and frequently by early dissolution. The commoner of these symptoms are precordial pain, irregularity or slowing of the cardiac rate, and convulsions.

The Aorta

The aorta, the principal arterial trunk of the greater or general circulation, arises from the aortic orifice at the superior and posterior portion of the left ventricle near the center of the heart at the level of the third left costal cartilage. The course of the thoracic aorta roughly resembles a shepherd's crook; and the vessel is divided into three portions, the ascending aorta, the aortic arch, and the descending aorta. The ascending aorta, which is enveloped by the apical portion of the pericardium, is approximately one and one-eighth inches in diameter, presenting near its origin the sinuses of Valsalva. The ascending aorta is two inches long, and at the upper border of the second right costal cartilage becomes continuous with the arch of the aorta. The ascending aorta occupies a position one-fourth inch behind the posterior surface of the sternum, separated from this bone by the enveloping pericardium and the right pleural sac. Near its termination the ascending aorta presents a dilatation, the *sinus maximus* which renders the lumen of the vessel at this point fusiform or oval. An aneurysm of the vessel at this point upon rupture discharges its blood directly into the pericardium with speedily fatal result. Prior to rupture an aneurysm of this portion of the aorta is prone to compress the right auricle, superior vena cava, or pulmonary artery, leading to embarrassment of the circulation and engorgement of the superficial veins of the neck.

The *aortic arch* commences at the level of the upper border of the second right costal cartilage and arches upward, backward, and to the left behind the manubrium sterni and in front of the trachea just above its bifurcation. Thence the vessel crosses over the left bronchus and passes downward to the left side of the body of the fourth thoracic vertebra, where it becomes continuous with the descending thoracic aorta. In the superior mediastinum three large arterial trunks, the innominate, the left common carotid, and the left subclavian, spring from the convexity of the arch; while by its lower concave aspect it is connected with the pulmonary artery by a fibrous cord, the ligamentum arteriosum, the remnant of the obliterated ductus arteriosus of the fetus.

Aneurysmal dilatation of the posterior wall of the aortic arch is apt to compress the trachea, producing cough, stridor, hemoptysis or fatal hemorrhage in the event of rupture. Pressure may also be exerted upon the esophagus with the production of dysphagia or upon the thoracic duct, resulting in lymph stasis or chylothorax. Similarly aneurysm of the anterior aspect of the arch may cause sternal protrusion with boring pain beneath the sternum. Through irritation of the left recurrent laryngeal nerve, which winds around this portion of the vessel, laryngeal symptoms may be induced, and through disturbance of the sympathetic nervous system pupillary changes may occur.

The *descending aorta,* commencing at the left side of the lower border of the fourth thoracic vertebra, descends in the deep portion of the posterior mediastinum to the aortic orifice of the diaphragm, through which it passes in front of the body of the twelfth thoracic vertebra. Aneurysm of this portion of the aorta, which is not an uncommon disease, usually extends backward along the left side of the vertebral column, where it causes boring pain and absorption of the bodies of the vertebræ, resulting in scoliosis or lordosis. When developing upon the anterior wall of the vessel, an aneurysm exerts pressure upon the pericardium and heart, pushing these structures before it, and giving rise to physical signs which suggest cardiac hypertrophy or pericarditis with effusion. In either situation rupture may occur into the mediastinum or pleural cavity with fatal termination.

Congenital abnormalities of the aorta are not common, though occasionally the aortic valve may present an abnormality. This may consist of adhesions between the borders of the valve cusps, or fenestration of the segments; or in rarer instances the orifice may be represented by a narrow slit, the button-hole orifice of Corrigan.

The Pulmonary Artery

The pulmonary artery, springing from the base of the right ventricle at the conus arteriosus, lies anterior to all of the great vessels which are connected with the base of the heart. A short wide trunk, two inches in length and embraced by the right and left auricular appendages, the pulmonary artery passes upward and backward to reach the concavity of the aortic arch, where, in front of the bifurcation of the trachea, at the level of the fourth dorsal vertebra, it divides to form the right and left pulmonary arteries, which enter the roots of their respective lungs.

Opening into the left auricle are the four *pulmonary veins,* which return arterial blood from the lungs to the left heart, while emptying into the right auricle are the *superior vena cava,* returning venous blood from the upper portion of the general circulation, and the *inferior vena cava,* returning venous blood from the abdominal cavity and lower extremities.

TOPOGRAPHIC ANATOMY

The Heart.—The *base* of the heart, formed by the right and left auricles, corresponds to a line crossing the sternum obliquely from the lower border of the second left costal cartilage at a point one-half inch to the left of its junction with the sternum to the upper border of the third right costal cartilage at a point one inch beyond its sternal junction.

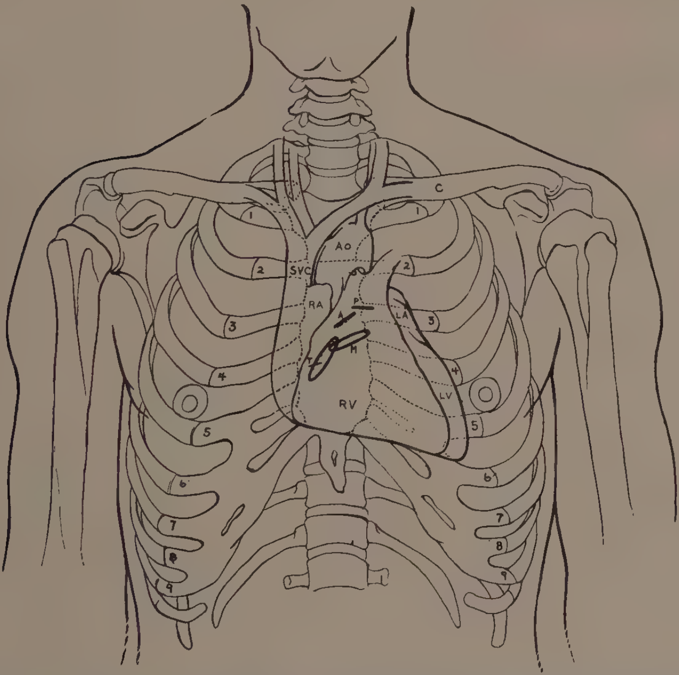


Fig. 11.—Relation of chambers of unopened heart to anterior thoracic wall. *LA,* left auricle; *LV,* left ventricle; *RA,* right auricle; *RV,* right ventricle; *P,* pulmonic valve; *A,* aortic valve; *M,* mitral valve; *T,* tricuspid valve; *AO,* aorta; *SVC,* superior vena cava.

The *right border* of the heart, formed by the right auricle, corresponds to a curved line, with its convexity directed toward the right, extending from the upper border of the third right costal cartilage one inch from its junction with the sternum, to the sixth right chondrosternal articulation.

The *inferior border* of the heart, formed almost entirely by the right ventricle, and to a minor extent by the left ventricle, corresponds to a line drawn from the sixth right chondrosternal articulation to the site of the normal cardiac impulse in the fifth left intercostal space one-half inch internal to the midclavicular line.

The *left border* of the heart, formed by the left ventricle, is represented by a curved line drawn with its convexity directed upward and toward the left, from the fifth left intercostal space one-half inch internal to the midclavicular line to the lower border of the second left costal cartilage, one-half inch to the left of its articulation with the sternum.

The *cardiac valves* all lie within a small ellipse extending from the third left chondrosternal articulation to the junction of the sixth right costal cartilage with the sternum. Within this elliptical area the *pulmonary valve* lies behind the third left chondrosternal articulation at the level of the upper border of the third costal cartilage. The *aortic valve* occupies a position behind the sternum a little distance to the right of the pulmonary valve at the level of the third intercostal space. The *mitral valve* lies behind the left sternal border at the level of the fourth costal cartilage. The *tricuspid valve* occupies a position behind the body of the sternum corresponding to the central point of a line drawn obliquely across the midsternal line from the third left to the sixth right chondrosternal articulation. In relation to the anterior wall of the thorax, the pulmonary valve is most superficial; the tricuspid is next in depth; the aortic more deeply placed than the tricuspid; while the mitral valve occupies the position most remote from the chest wall. This area overlies the anatomic site of the valves, and is not the area in which the sounds produced at the several valves are best appreciated acoustically.

The Aorta.—The ascending portion of the aorta is represented on the surface of the thorax by a broad line drawn from the third left chondrosternal junction to the upper border of the second right costal cartilage at its junction with the sternum. From this point the arch of the aorta takes a course backward and toward the left behind the manubrium sterni, its convexity ascending to a point one inch below the episternal notch, its concavity corresponding to the level of the angle of Louis.

The *pulmonary artery* corresponds to a broad line drawn from the second left intercostal space at the sternal border to the upper border of the second left costal cartilage, the point of bifurcation of the artery to form its two primary branches.

The Precordia.—The term precordia is applied to the area of the surface of the thorax which overlies the heart and pericardium. The name does not refer merely to the limited region in which the heart is directly in apposition with the thoracic wall, but also to the region in which the anterior borders of the lungs overlap the pericardium and heart. The region embraces the areas of relative cardiac dullness and cardiac flatness, to be described in a subsequent section. The precordia presents sharp lines of demarkation superiorly and upon the left; but upon the right side and inferiorly it is continuous with the areas of hepatic dullnes and flatness.

CHAPTER III

CLINICAL ANATOMY AND PHYSIOLOGY OF THE ABDOMEN

The abdomen is that portion of the trunk which is limited upon the surface of the body by the ensiform cartilage and the costal arch superiorly and by the pubic crest and Poupart's ligaments inferiorly. It presents an irregularly oval contour, the shape varying, however, with the age and the sex of the subject. Thus, the abdomen of the child is roughly conical with the apex directed inferiorly; while in the adult female subject, owing to the unusual breadth of the pelvis in this sex, it is roughly conical with the apex directed superiorly; whereas in the case of the adult male subject the abdomen is oval or barrel-shaped, with a moderate anteroposterior flattening.

The *abdominal cavity* is limited superiorly by the inferior aspect of the diaphragm, and inferiorly by the levator ani, assisted by the coccygeus, these two muscles constituting the pelvic diaphragm. The more roomy upper portion of the abdominal cavity, situated above the brim of the pelvis, is termed the *abdomen proper,* while the smaller portion of the cavity situated below the pelvic inlet is termed the *pelvis.*

The abdominal cavity is not limited superiorly by the lower border of the costal arch, which forms its upper boundary upon the surface of the abdomen, but by the vault of the diaphragm, extending upward into the bony thorax for a considerable distance. Upon the right side its superior limit is on a level with the upper border of the fifth rib in the midclavicular line; while upon the left side of the body its superior limit is approximately one-half inch lower in the same line.

The *abdominal wall* is composed largely of muscular and soft structures, reinforced in certain regions by bony structures. Anteriorly and laterally the wall is formed of the abdominal muscles, the lower ribs and iliac bones. Posteriorly it is formed by the muscles of the posterior abdominal wall, the quadratus lumborum and psoas upon either side, and in the median line by the vertebral column. The anterior abdominal wall and the lateral walls between the last rib and the iliac crest are devoid of bony support and are subject to distention and retraction, depending upon the state of the abdominal contents.

Within the abdominal cavity the liver occupies the upper right quadrant of the cavity, sheltered largely beneath the inferior right costal margin, but extending also into the subcostal angle in the epigastric region. The spleen occupies a deep position in the upper left quadrant, sheltered by the ninth, tenth, and eleventh ribs and in apposition with the fundus of the stomach. The stomach occupies a position in the superior portion of the abdominal cavity, between the liver and the spleen; and from the greater curvature of the stomach the great omentum descends for a variable distance and covers the coils of the small intestine. The coils of the small intestine occupy the central and inferior portions of the abdominal cavity, slightly overlapping the ascending and descending colon in the lumbar regions. The large intestine envelops the small intestine in the form of a frame with an inferior concavity upon three sides of the abdominal cavity. The urinary bladder occupies the anterior portion of the pelvis; and, if it is in a state of distention, rises above the pubic crest to mount into the hypogastric region. The rectum occupies the posterior portion of the pelvis, lying in the concavity of the sacrum.

THE STOMACH

The position of the stomach in the abdominal cavity varies with the habitus of the individual. In the subject of normal habitus the stomach occupies the left hypochondriac and epigastric regions of the abdomen. In such a subject the *cardiac orifice* of the stomach is located behind the seventh left costal cartilage, at a point one inch from the sternum. The position of the *pyloric orifice* is somewhat variable, its site being modified by the state of the stomach. In the fasting state this orifice occupies a position in the median line of the abdomen, at a point midway between the episternal notch and the symphysis pubis. When the stomach is moderately distended, the pylorus occupies a position approximately one inch to the right of the median line at the same level, this displacement to the right being increased to two or three inches in the presence of extreme distention of the organ.

The *fundus* of the stomach is in contact with the inferior aspect of the diaphragm, behind and below the apex of the heart, in which position it extends as high as the sixth rib. The *lesser* *curvature,* representing the superior limit of the stomach, is covered by the left lobe of the liver, passing downward and toward the right from the cardiac orifice to the pyloric orifice of the stomach. The *greater curvature,* representing the inferior limit of the organ, crosses the left costal arch at the level of the ninth left costal cartilage, the most dependent portion of the normal organ occupying a level approximately two inches above the umbilicus.

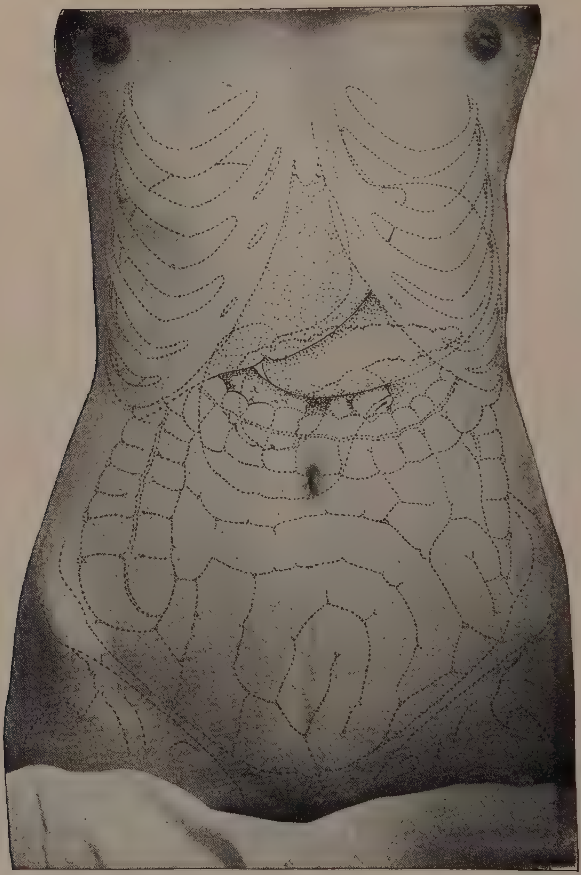


Fig. 12.—The central upper abdomen. Showing in outline the liver and stomach and pancreas. (From Crossen.)

The *anterior surface* of the stomach, largely overlapped by the left lobe of the liver, inferior border of the left lung and left costal arch, is exposed to the anterior abdominal wall in a very limited portion of its extent. *Traube's semilunar space,* the area in which the anterior wall of the stomach is in direct contact with the anterior abdominal wall, affording upon percussion pure gastric tympany, is limited superiorly and upon the right by the left lobe of the liver, superiorly and upon the left by the inferior border of the left lung, and inferiorly by the transverse colon.

The *posterior surface* of the stomach is directed downward and backward, reposing in the so-called "stomach bed," formed by the transverse mesocolon, the pancreas, left kidney, and suprarenal capsule.

In the subject of asthenic habitus, characterized by acute costal angle and long thorax, the stomach occupies largely a vertical position along the left side of the vertebral column, and the greater curvature may occupy a position below the umbilicus or even below the iliac crests.

In the subject of hypersthenic habitus, on the contrary, in whom the thorax is short and the costal angle obtuse, the stomach occupies a position high in the abdomen and pursues an oblique course across the vertebral column in the epigastric region.

THE SPLEEN

The spleen is a solid organ situated deeply in the left hypochondriac region. The organ occupies an oblique position in the abdominal cavity, between the fundus of the stomach, the left kidney, and the diaphragm, its long axis corresponding to the course of the tenth rib. Owing to its accurate apposition with the inferior aspect of the diaphragm, the spleen exhibits respiratory mobility, and also a certain range of mobility with changes in the bodily posture.

While the normal spleen is subject to certain variations in form, its usual contour is elongated with a considerable degree of flattening; and when the spleen assumes this form it presents three surfaces; namely, a diaphragmatic surface, a gastric surface, and a less extensive renal surface. The diaphragmatic surface of the spleen is closely applied to the inferior surface of the left vault of the diaphragm, which separates the organ from the pleural cavity and the lower border of the left lung between the ninth, tenth, and eleventh ribs. The gastric surface of the spleen, directed forward, inward, and downward, is in relation with the fundus of the stomach. The renal surface of the spleen, directed inward and downward, is the least extensive of the three surfaces of the organ. It is in relation with the outer aspect of the superior pole of the left kidney and occasionally with a portion of the left suprarenal capsule.

The superior or posterior extremity of the spleen is on a level with the eleventh thoracic vertebra, and occupies a position one and one-half inches from the midspinal line. The inferior or anterior extremity of the organ reaches the midaxillary line, and occasionally the anterior axillary line in the normal subject. The anterior extremity of the spleen commonly reaches as far forward as the *costoarticular line*, which is a diagonal line erected

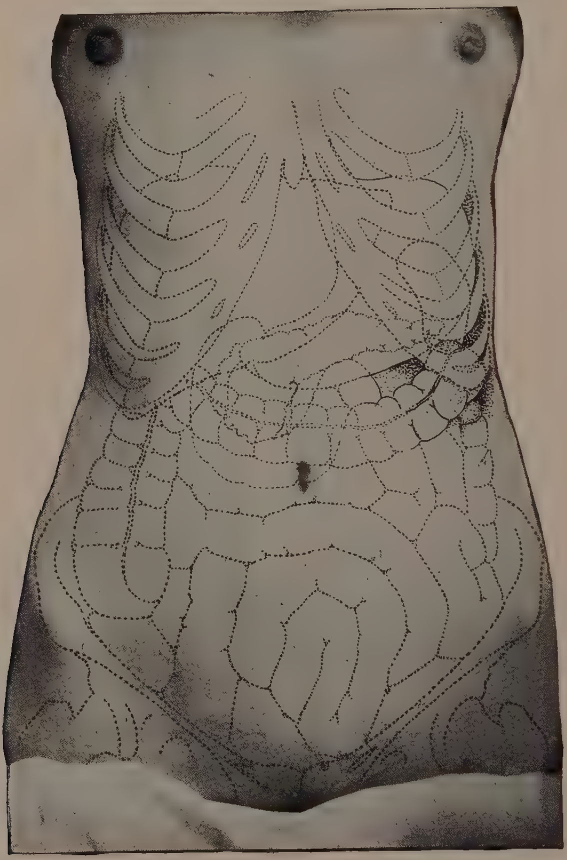


Fig. 13.—The left upper abdomen. The site of the spleen and of the splenic flexure of the colon, the organs in this region most commonly affected, are shown by the stippling. When normal, the spleen lies considerably higher in the abdominal cavity than is generally supposed. Its anterior projection is shown here in dotted outline, with the lower end in contact with the splenic flexure of the colon. (From Crossen.)

upon the anterolateral wall of the trunk from the anterior extremity of the eleventh left rib to the left sternoclavicular articulation. Projection of the anterior extremity of the spleen beyond this line is indicative of enlargement or displacement of the organ.

The spleen is maintained in its normal habitat through the medium of the splenic ligaments, composed of folds of peritoneum, and by the intraabdominal tension exercised upon its inferior surface, and counterbalanced by the intrathoracic tension exercised upon the superior aspect of the diaphragm.

The spleen is invested by a fibrous capsule and by a superjacent investment of peritoneum, save at the hilum of the organ situated upon the margin intervening between the gastric and the renal surfaces of the organ, at which point the splenic vessels enter and leave the organ. In the presence of inflammation of the organ the peritoneal coat frequently becomes roughened and rugose, with the production of the peritoneal friction sounds of Beatty-Bright. Moreover, as the splenic vein forms one of the principal tributaries of the portal vein, in the presence of valvular disease of the heart, or in portal obstruction from hepatic cirrhosis, the spleen becomes the seat of passive congestion with enlargement of the organ.

The size and weight of the spleen present numerous variations at different periods of life and under varying circumstances in the adult subject. The normal adult spleen is approximately five inches in length, three inches in breadth, and one inch in thickness. The spleen exhibits a moderate increase in volume during digestion; and the organ is regularly of ample dimensions in the well nourished subject, to become very small during starvation.

The weight of the spleen in proportion to that of the entire body is approximately the same at birth as it is in the case of the adult subject. At birth the proportion is 1:350, while in the healthy adult subject it is 1:320 to 1:400. With advancing age, however, the organ not only decreases considerably in weight, but even decreases considerably in proportion to the general weight of the body, frequently falling to the proportion of 1:700.

Occasionally in the healthy adult the spleen possesses a roughly tetrahedral form, while in other instances it is irregularly quadrilateral, with the result that instead of the long axis of the organ conforming to the course of the tenth rib, it occupies a plane perpendicular to this rib.

The spleen commonly corresponds to an area upon the surface of the body extending from the upper border of the ninth rib to the lower border of the eleventh rib, its inner extremity being situated one and one-half inches from the midspinal line, and its anterior extremity reaching as far forward as the midaxillary or the anterior axillary line. Superiorly the area of splenic dullness is limited by the inferior border of pulmonary resonance of the left lung, while inferiorly it is continuous with the area of renal dullness.

THE KIDNEYS

The kidneys are solid organs situated in the posterior portion of the abdomen, behind the peritoneum, upon either side of the vertebral column. The superior extremities of the kidneys correspond to the level of the upper border of the twelfth dorsal vertebra; while the inferior extremities are on a level with the third lumbar vertebra. Owing to its relation with the right lobe of the liver, the right kidney occupies a slightly lower level in the abdominal cavity than does the left kidney.

The normal adult kidney is approximately four and one-half inches in length, two and one-half inches in width, and two inches in thickness. The left kidney is slightly longer and narrower than is the right kidney. In the infant and the young child the kidneys are relatively larger than in the adult subject. The relative weight of the kidney in the adult to the entire body weight is 1:240, whereas in the infant the proportion is 1:120.

Posteriorly the kidneys rest upon the muscles of the posterior abdominal wall, the psoas, the quadratus lumborum, the fascia of the transversalis, and the diaphragm. The anterior surface of the right kidney is in relation with the inferior aspect of the liver, the descending portion of the duodenum and the hepatic flexure of the colon and the adrenal. The large intestine is united to the anterior face of the kidney by areolar tissue, with the result that in the presence of enlargement of the kidney from neoplasm or other cause the large intestine is carried before the kidney in its hypertrophy. The anterior surface of the left kidney is in relation with the posterior surface of the stomach, the spleen, the body of the pancreas, the jejunum, the adrenal and the splenic flexure of the colon. While the large intestine passes across the ventral face of the left kidney, it is not moored securely to the kidney, with the result that with enlargement of the kidney the colon is occasionally but not invariably carried in front of the organ.

The kidneys occupy portions of the epigastric, umbilical, hypochondriac, and lumbar regions upon either side of the median line. The superior pole extends as high in the epigastric region as a transverse line drawn about two inches below the ensiform process. The inferior pole extends below the subcostal line, only slightly in the case of the left kidney, and to a greater degree in

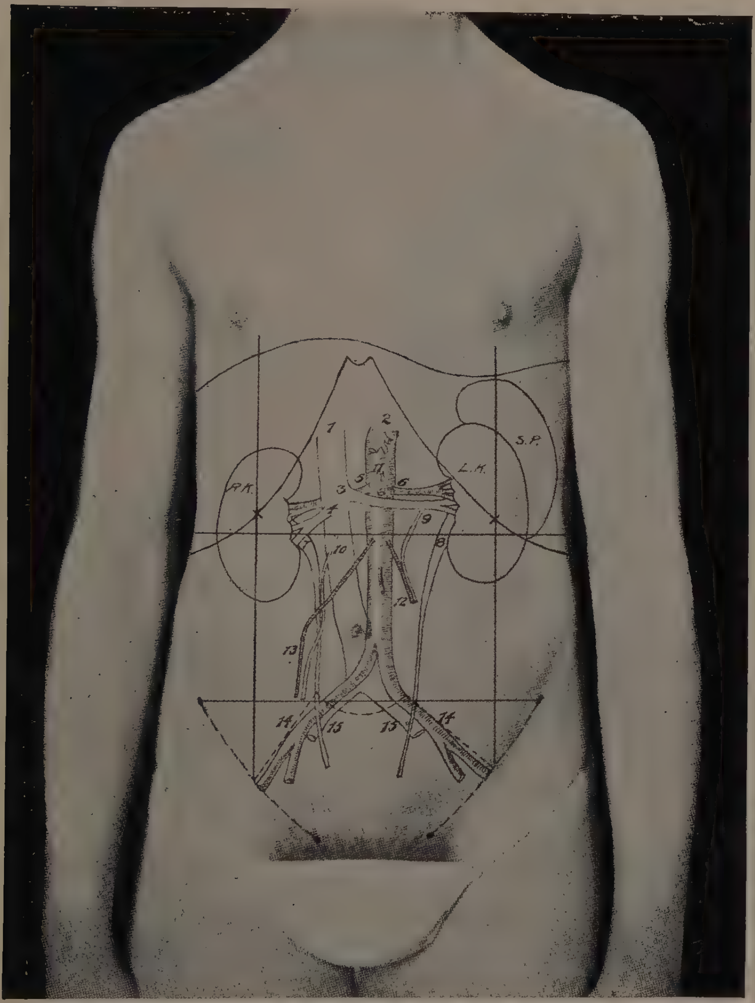


Fig. 14.—Surface markings of kidneys, ureters and abdominal vessels. Anterior view. (From Eisendrath.)

1, inferior vena cava; *2,* aorta—celiac axis just below *2*; *3* and *4,* right and left renal veins; *5* and *6,* right and left renal arteries; *7* and *8,* right and left ureters; *9,* left spermatic vein; *10,* right spermatic vein; *11,* superior mesenteric artery; *12* and *13,* right and left spermatic arteries; *14,* external iliac arteries; *15,* external iliac veins; *RK,* right kidney; *LK,* left kidney; *SP,* spleen.

the case of the right kidney. In the female subject the kidneys occupy a lower position in the abdominal cavity than in the male subject. In both sexes the superior pole of the organ is nearer the median line of the body than is the inferior pole.

The kidney is roughly reniform in shape, presenting a convex external border which conforms anatomically to the outer border of the quadratus lumborum muscle, and presenting a concave internal border, which bears in its midportion a deep fissure, the hilum of the kidney, for the entrance and exit of the renal vessels, nerves, lymphatics, and the ureters.

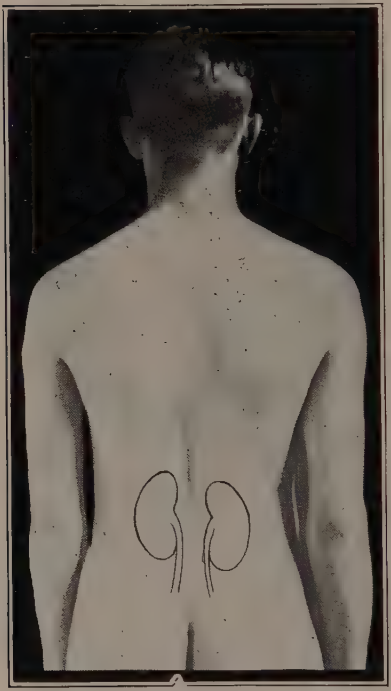


Fig. 15-*A*.—Topographic anatomy of kidneys and ureters.

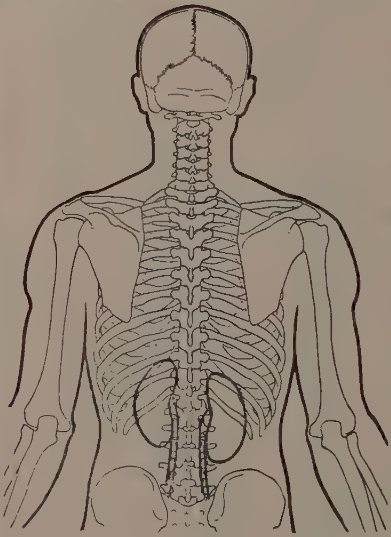


Fig. 15-*B*.—Topographic anatomy of kidneys and ureters.

The kidney is maintained in position by a surrounding bed of adipose tissue, the perirenal fat, and by a sheath of fibrous tissue, which is continuous in the median line of the abdominal cavity and which is anchored superiorly with the fascia of the diaphragm.

Congenital absence of one kidney has been observed, and not infrequently one kidney is considerably larger than its fellow of the opposite side. Occasionally also the kidneys are fused at their inferior poles with the production of a "horseshoe kidney." In other instances the fusion of the organs is complete, the kidneys being represented by a single large organ with two ureters.

THE LIVER AND GALL BLADDER

The liver, the largest gland of the body, occupies the upper right quadrant of the abdominal cavity, lying in the right hypochondriac region, the larger portion of the epigastric region, the thin extremity of the left lobe extending into the left hypochondriac region, and the inferior extremity of the right lobe invading the right lumbar region for a short distance. The liver is roughly wedge-shaped, with the wide base directed toward the right, and the thin sharp edge of the wedge directed toward the left side of the abdomen. The normal adult liver measures eight to nine inches transversely, six to seven inches vertically at the base of the wedge, and four to five inches anteroposteriorly at a point on a level with the upper border of the right kidney.

The liver is divided into two unequal portions, the right and left lobes, by the falciform ligament and longitudinal fissure, the right lobe greatly exceeding the left lobe in size. At the point where the falciform ligament joins the inferior margin of the liver there is a small notch, the umbilical notch, which is situated at the level of the ninth right costal cartilage one inch to the right of the median line. Slightly beyond this notch the liver presents a second notch, in which is lodged the fundus of the gall bladder, corresponding to the junction of the ninth rib and the right border of the rectus muscle.

The *superior surface* of the liver, smooth and convex, is closely applied to the concave right vault of the diaphragm. Upon its central portion the superior surface of the liver presents a shallow depression, the *cardiac depression*, corresponding to the position of the heart upon the superior surface of the diaphragm.

The *anterior surface* of the liver is applied to the inferior surface of the diaphragm, which separates it from the lower ribs and their cartilages upon the right and left sides, while in the median line it comes into direct contact with the anterior abdominal wall in the subcostal angle.

The *right* and *posterior* surfaces of the liver are in contact with the inferior aspect of the diaphragm, which separates the liver from the right pleural cavity and the lower border of the right lung.

The *inferior surface* of the liver, directed somewhat posteriorly and toward the left, is in relation with the stomach, the hepatic flexure of the colon, the right kidney and suprarenal capsule, the second portion of the duodenum and the gall bladder.

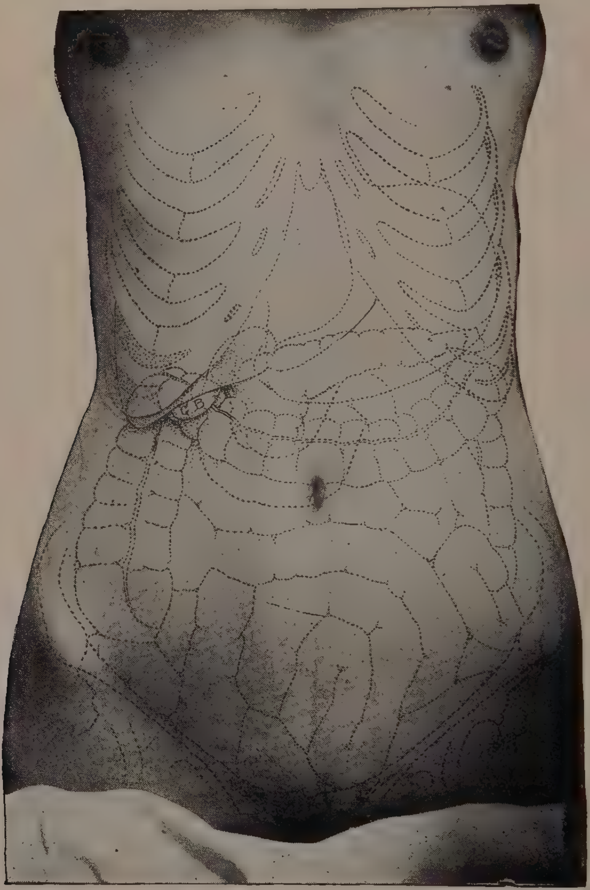


Fig. 16.—The right upper abdomen. The site of the gall bladder, the area of particular interest in this region, is indicated by the letters *G.B.* (From Crossen.)

The hepatic parenchyma is enclosed in a fibrous capsule, which is in turn invested with peritoneum with the exception of a limited portion of the posterior surface of the liver, constituting the "bare area" of the organ, which is united to the inferior surface of the diaphragm by areolar tissue. During the course of inflammatory disease of the organ the serous investment occasionally becomes rugose and roughened with the production of a peritoneal friction sound upon the respiratory movements of the organ or upon manipulation of the abdominal wall in the hepatic region.

In addition to the coronary ligament, the liver is retained in position by the falciform ligament, the round ligament, and two lateral ligaments. The falciform ligament is a broad fold of peritoneum which is attached on the one hand to the inferior surface of the liver at the umbilical notch, and upon the other hand to the inferior surface of the diaphragm and to the posterior surface of the sheath of the rectus muscle as low as the umbilicus. The round ligament is a small fibrous cord representing the remnants of the umbilical vein after it has undergone occlusion. It passes from the umbilicus in the free margin of the falciform ligament to the umbilical notch of the liver, whence it passes along the umbilical fissure upon the inferior surface of the liver, to be continued upon the posterior surface of the organ as the impervious ductus venosus up to the inferior vena cava. The lateral ligaments attach the extremities of the liver to the inferior surface of the diaphragm, being largely composed of the lateral reflections of the coronary ligament.

The liver is supported in the abdominal cavity partially by virtue of its ligamentous connections with the abdominal walls, partially by an areolar attachment to the inferior aspect of the diaphragm in the "bare area" of the posterior surface of the organ, and partially by the pressure which is exerted upon the organ by the other abdominal viscera. In the event of relaxation of the hepatic ligaments, the liver falls downward to occupy a lower level in the abdominal cavity; and in the presence of general visceroptosis the liver shares in the descent of the abdominal viscera.

The liver is movable within certain limits. Owing to its accurate apposition with the inferior surface of the diaphragm, it participates in the respiratory excursions of this muscle, descending during inspiration and ascending during expiration. The weight of a right-sided pleural effusion or the pressure exerted in right pneumothorax is equally capable of depressing the liver and causing its sharp inferior border to project below the right costal arch.

The habitual wearing of tight clothing exercises a distinct influence upon the form and the position of the liver. There is under these circumstances a progressive flattening of the superior surface of the organ with a concomitant lengthening of the right lateral surface upon which is frequently noted a constriction corresponding to the edge of the costal arch, the tapering lower extremity of the right lobe in this state constituting Riedel's lobe.

The portal vein and hepatic artery enter the transverse fissure of the liver between the layers of the gastrohepatic omentum, which unites the liver with the stomach. The hepatic duct, in close relation with these vessels, passes downward from the transverse fissure between the layers of the same omentum.

The *gall bladder,* the reservoir for the bile, reposes in a shallow depression upon the inferior surface of the right lobe of the liver, the tip of the fundus meeting the anterior abdominal wall in the notch upon the inferior border of the liver situated at the junction of the ninth right costal cartilage with the outer border of the right rectus muscle.

The gall bladder is a pear-shaped sac which is approximately four inches in length and one and one-half inches in diameter when moderately distended. From the fundus of the sac, the body tapers to form the neck, which in turn terminates in the cystic duct, which unites with the hepatic duct to form the common bile duct.

The common bile duct, the common excretory duct of the liver and the gall bladder is approximately three inches in length. It descends in the folds of the lesser omentum, passing behind the first portion of the duodenum, and passing between the head of the pancreas and the descending portion of the duodenum, it empties its contents into the second portion of the duodenum usually by a common orifice with the pancreatic duct. As a result of the relations of the common bile duct to the pancreatic head and the descending portion of the duodenum, stenosis of the duct frequently ensues in the event of malignant disease of the head of the pancreas with the production of hepatic enlargement and icterus. Stenosis of the duct may also ensue as the result of the lodgment of gallstones along its course. In the event of the impaction of a large gallstone at the common orifice of the common bile duct and of the pancreatic duct, it has been demonstrated that the pressure of the bile in the common duct is capable of overcoming the pressure in the pancreatic duct, with the result that bile accumulates in the pancreatic duct, perhaps with the production of inflammation of the pancreas.

Surface Topography.—The *superior border* of the liver corresponds to the level of the lower border of the sixth rib in the midclavicular line, the lower border of the eighth rib in the midaxillary line, and the lower border of the tenth rib in the scapular line.

The *inferior border* of the liver corresponds to a line drawn downward and toward the right from the lower border of the sixth rib in the left midclavicular line, the point upon the surface corresponding to the left extremity of the organ, the line crossing the left costal arch at the eighth costal cartilage, the median line four inches below the ensiform cartilage, the right costal arch at the ninth costal cartilage, the lower border of the tenth rib in the midaxillary line, and the lower border of the eleventh rib in the scapular line.

THE PANCREAS

The pancreas is an elongated, solid organ comprising a head, neck, body, and tail, situated deeply in the epigastric region, its tail, however, extending into the left hypochondriac region.

The pancreas in the adult subject is approximately six inches in length, and its location in the abdominal cavity corresponds to the level of the first and second lumbar vertebræ. The head of the pancreas is encircled by the second and third portions of the duodenum, the common bile duct intervening between the two structures, while the tail extends toward the left and is in relation with the hilum of the spleen.

The body of the pancreas is roughly prismatic in shape and possesses three surfaces, which respectively are directed anteriorly, posteriorly, and inferiorly. The anterior surface, covered by the peritoneum of the lesser sac, is in relation with the posterior wall of the stomach, the transverse colon, and a few coils of the small intestine. The posterior surface, devoid of peritoneal covering, rests upon the abdominal aorta, the inferior vena cava, the right crus of the diaphragm, the splenic vein, the left kidney, renal vessels, and suprarenal capsule, and the commencement of the superior mesenteric artery. The posterior aspect of the neck of the pancreas is in relation with the commencement of the portal vein. The inferior surface is in relation with the duodenojejunal flexure of the small intestine, a few coils of the jejunum, and its left extremity rests upon the splenic flexure of the colon. As a result of the intimate relation of the pancreas to so many important blood vessels, enlargements of the gland are not infrequently attended by vascular bruits due to narrowing of the lumen of one or more of these vascular trunks. Similarly, in malignant disease of the head of the pancreas, the common bile duct, intervening between this portion of the gland and the second portion of the duodenum is frequently compressed with the production of icterus, which may simulate that of hepatic disease.

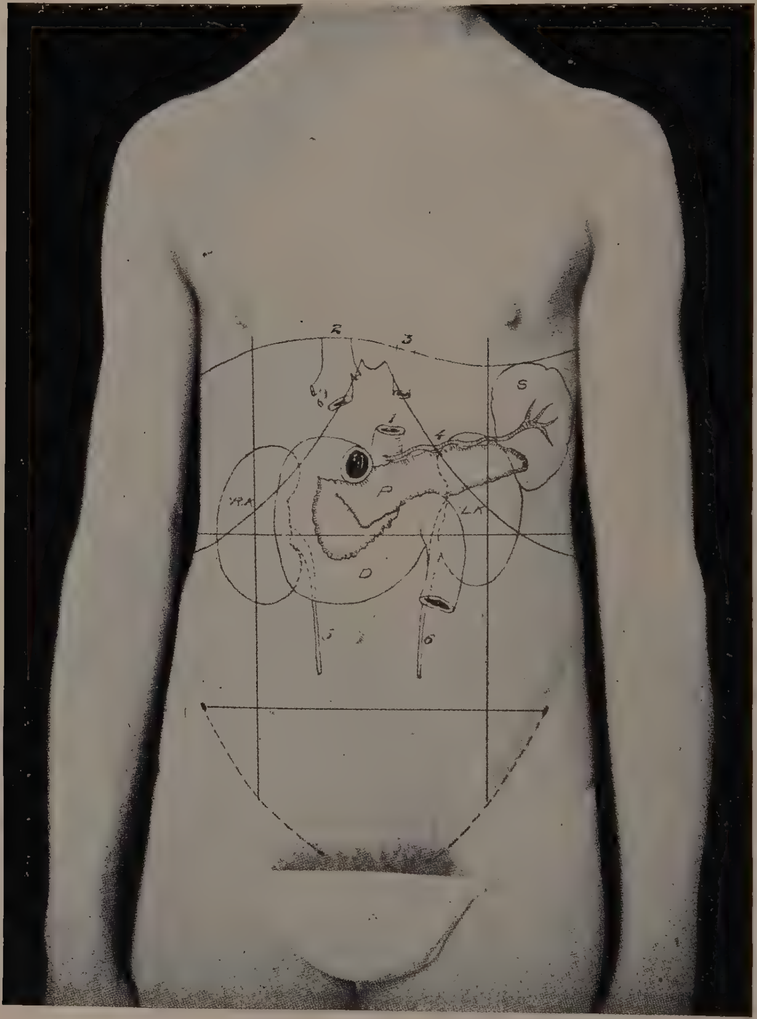


Fig. 17.—Relations of pancreas to adjacent viscera.

1, aorta; *2*, inferior vena cava; *3*, esophagus; *4*, splenic artery; *5* and *6*, ureters; *RK*, right kidney; *LK*, left kidney; *D*, duodenum; *P*, pancreas; *S*, spleen. (From Eisendrath.)

The pancreatic secretion is conveyed to the second portion of the duodenum by the pancreatic duct of Wirsung, which empties its contents into this portion of the intestine by a common orifice with the common bile duct. Occasionally the pancreas possesses an accessory excretory duct, the duct of Santorini, which empties its contents into the second portion of the duodenum approximately one inch above the level of the orifice of the principal pancreatic duct. Stenosis of the pancreatic duct usually leads to cystic enlargement of the pancreas; and the entrance of infective bacteria from the intestine by way of the pancreatic duct may induce suppurative disease of the pancreas. Malignant disease of the pancreas usually involves the head of the gland to the exclusion of other portions, and carcinoma comes first in frequency among pancreatic morbid growths, although sarcoma and adenoma also attack the gland.

Malignant growths of the head of the pancreas are prone to result in complete obstruction of the common bile duct as it passes between the head of the gland and the descending duodenum, resulting in deep obstructive jaundice.

The pancreas crosses the lower portion of the epigastric region, the tail of the gland extending approximately two inches into the left hypochondriac region, and the lower extremity of the head of the gland dipping into the upper portion of the umbilical region. The lowest point of the gland occupies a position approximately three inches above the umbilicus.

THE SMALL INTESTINE

The small intestine, the section of the gastrointestinal tract extending from the pyloric orifice of the stomach to the junction with the large intestine at the ileocecal valve, lies within the frame formed by the course of the large intestine, slightly overlapping the ascending and the descending colon, and extending for a variable distance below the brim of the pelvis.

The *duodenum,* the proximal twelve inches of the small intestine, pursues a course resembling the letter ''C'' from the pylorus to the duodenojejunal flexure at the left side of the second lumbar vertebra, embracing in its course the head of the pancreas and the common bile duct, which empties its contents into this portion of the small intestine.

Duodenal ulcer is the most important lesion of this portion of the small intestine. In the majority of cases the ulcer involves the anterior wall of the duodenum within three-fourths of an inch of the pylorus. The usual site of tenderness in duodenal ulcer is upon the anterior abdominal wall approximately four finger-breadths above the umbilicus and midway between the right sternal and parasternal line.

The duodenum is only rarely the seat of carcinoma. Maydl in analyzing 41,838 cases of carcinoma found at autopsy in the Pathologic Institute of Vienna, found the gastrointestinal tract involved in 10 per cent, and in these the duodenum was involved in only 7 instances, as against 10 in the ileum, 164 in the colon, and 162 in the rectum.

The *jejunum,* the second division of the small intestine, comprising approximately eight feet of the tube, lies in the umbilical and right and left lumbar regions, and is freely movable.

The *ileum,* the distal twelve feet of the small intestine, which terminates at the ileocecal valve, lies in the umbilical, hypogastric, and lumbar regions, in which it is freely movable. Only the great omentum intervenes between the jejunum and ileum and the anterior abdominal wall.

THE LARGE INTESTINE

The large intestine, comprising the terminal six feet of the gastrointestinal canal, comprises the cecum with the vermiform appendix, the ascending, transverse, and descending colon, the sigmoid flexure and the rectum. The large intestine is arranged in the form of a frame enclosing the coils of the small intestine upon the right side, superiorly, and upon the left side of the abdominal cavity, the ascending colon and the descending colon being slightly overlapped by the coils of the small intestine.

The *cecum,* with the vermiform appendix, occupies the right iliac and the hypogastric regions. The cecum, comprising the blind extremity of the great intestine, lies inferiorly to the ileocecal valve, is approximately three inches in diameter and two and one-half inches in length. The *vermiform appendix* is a slender appendage of the cecum, usually approximately four inches in length and one-fourth inch in diameter, which is commonly given off from the postero-internal surface of the cecum. The appendix is quite free and mobile, and while it frequently inclines downward toward the brim of the true pelvis, it may be deflected in other directions.

The *ascending colon* ascends vertically in the right lumbar and right hypochondriac regions in contact with the anterior wall of the abdomen to reach the inferior surface of the liver at the level of the tenth right costal cartilage. At this level the colon is flexed toward the left and downward, forming the hepatic flexure, which terminates in the transverse colon. The ascending colon is approximately eight inches in length; and in its ascent it is in relation with the anterior surface of the right kidney, and it terminates in the hepatic flexure at a point upon the inferior surface of the liver which is situated external to the gall bladder.

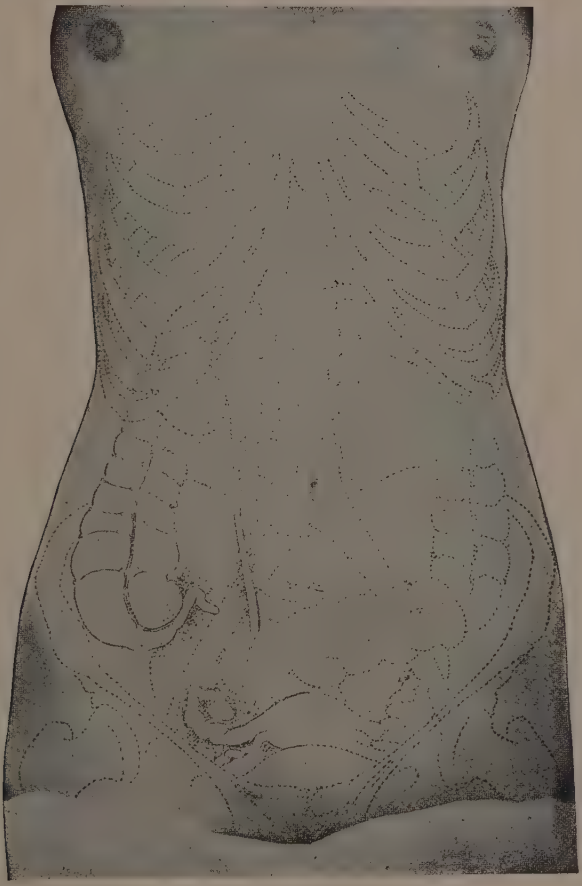


Fig. 18.—The right lower abdomen. The organs commonly affected, and the areas accordingly of particular interest, are indicated by the stippling. (From Crossen.)

The *transverse colon* crosses the abdominal cavity, descending from the right hypochondriac region into the upper portion of the umbilical region; thence ascending obliquely into the left hypochondriac region to the inferior extremity of the spleen to form the splenic flexure of the colon. The transverse colon, approximately twenty inches in length, is attached to the posterior abdominal wall by a broad fold of peritoneum, the transverse mesocolon, and it is the most freely mobile portion of the large intestine. Anteriorly the transverse colon is in apposition with the anterior abdominal wall, the great omentum alone intervening between the two structures. Posteriorly the transverse colon is in relation with the second or descending portion of the duodenum, and with coils of the small intestine. The transverse colon and transverse mesocolon serve somewhat as a dividing factor in the abdominal cavity, the liver, gall bladder, stomach, and spleen lying above this portion of the great intestine; and the coils of the small intestine occupying a position below its level.

The *descending colon,* commencing at the splenic flexure, passes vertically downward in the left lumbar and left iliac regions, to terminate in the sigmoid flexure in the hypogastric region. The descending colon is approximately nine inches in length; it is in relation anteriorly and laterally with the abdominal walls; and posteriorly it is in relation with the outer border of the left kidney and with the muscles forming the posterior abdominal wall.

The *sigmoid flexure* is approximately thirteen inches in length, commencing at the level of the iliac crest and terminating at the brim of the true pelvis in the rectum opposite the left sacroiliac joint. The sigmoid flexure is the narrowest portion of the large intestine. When empty and collapsed, it falls into the rectovesical or rectovaginal pouch; but when distended with fecal accumulation or with gas it mounts into the abdominal cavity, occasionally reaching the level of the umbilicus. The sigmoid flexure is attached to the posterior abdominal wall by a fold of peritoneum, the mesosigmoid, which permits this portion of the large intestine to enjoy a considerable degree of mobility. As a result of this free mobility the sigmoid flexure is occasionally the site of volvulus, an accident which is favored in many cases by elongation of the mesosigmoid from the weight of accumulation of fecal material in chronic constipation.

The *rectum,* the terminal portion of the large intestine, is approximately nine inches in length. Commencing at the brim of the true pelvis at the left sacroiliac joint, it passes obliquely downward and inward to reach the third sacral joint, whence it continues its course directly downward in the median line of the pelvis to a point one inch below the tip of the coccyx, where it turns abruptly backward to form the anal canal.

THE URETERS AND BLADDER

The ureters are cylindrical membranous tubes from ten to twelve inches in length and one-fourth inch in diameter which take their origin from the pelves of the kidneys and terminate in the walls of the urinary bladder. The ureters pursue an oblique course downward and inward through the abdominal cavity, to cross the brim of the pelvis and enter the walls of the bladder. The course of the ureter is indicated upon the surface of the abdomen by a line drawn almost vertically from a point in the umbilical region approximately five cm. external to the median line of the body, at a level of the anterior extremity of the twelfth rib, to a point a little below the umbilicus, whence the course of the tubes converge toward the median line as the symphysis pubis is approached.

The urinary bladder is a hollow viscus lying posterior to the symphysis pubis. The empty viscus is entirely within the pelvis, but when distended may reach as high as the umbilicus. The walls of the bladder are composed of unstriped muscle and connective tissue. The inner surface is lined with mucous membrane which is thrown into folds when the bladder is empty.

THE OVARIES, UTERUS AND FALLOPIAN TUBES

The ovaries are located below the brim of the female pelvis about five cm. from the symphysis pubis. They vary considerably in size, the average being about three-fourths by one-half inch, increasing in size somewhat during ovulation.

The uterus occupies a position in the female pelvis posterior to the bladder and anterior to the rectum. In the adult the non-pregnant uterus measures about two and one-half inches in length. During pregnancy, however, the dome of the uterus may reach as high as the ensiform cartilage.

The fallopian tubes extend laterally from the body of the uterus, the fimbrial extremities terminating at the ovaries.

Clinical anatomy of the external genitals for the purpose of physical diagnosis is identical with the gross anatomy of the organs, and should be familiar to students or reviewed from a textbook on the subject.

ANATOMIC LANDMARKS OF THE ABDOMEN

At the superior limit of the anterior abdominal surface in the median line is the *ensiform cartilage,* with the *costal arch* descending from it upon either side of the abdomen. The anterior ex-

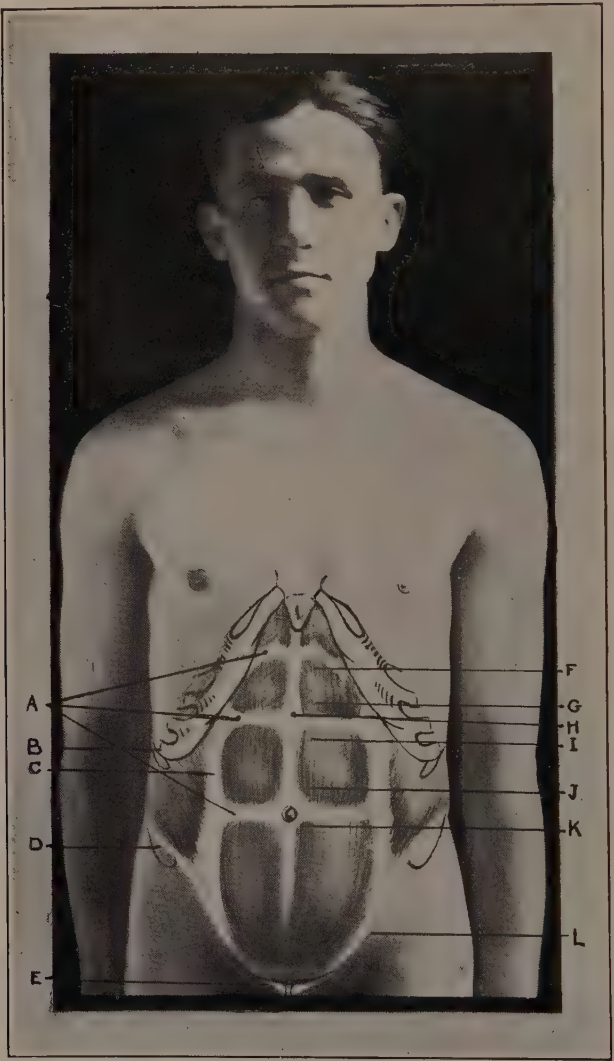


Fig. 19.—Anatomic landmarks of abdomen:

A. Lineæ transversæ. B. Costal arch. C. Linea Semilunaris. D. Anterior superior iliac spine. E. Symphysis pubis. F. Commencement of abdominal aorta. G. Origin of celiac axis. H. Linea alba. I. Origin of superior mesenteric artery. J. Origin of inferior mesenteric artery. K. Bifurcation of aorta. L. Poupart's ligament.

tremities of the fifth, sixth, seventh, eighth, ninth, and tenth costal cartilages are palpable; and, in the case of thin subjects with relaxation of the abdominal walls, the free extremities of the eleventh and twelfth ribs as well may be palpated.

At the inferior limit of the abdomen the *symphysis pubis* with its *pubic spines* is encountered, and extending from them in a direction outward and obliquely upward, one encounters *Poupart's ligament* upon either side of the abdomen.

In the inferior and lateral regions the *iliac crest*, terminating anteriorly in the *anterior superior iliac spine* is encountered, the latter remaining plainly palpable even in obese subjects.

In the lower central region of the abdominal surface the *umbilicus* is noted. It corresponds to the level of the disc between the third and fourth lumbar vertebræ.

The *linea alba* extends in the median line from the ensiform cartilage to the symphysis pubis. It is indicated by a slight groove in the median line of the abdomen above the level of the umbilicus, and by a line of hair or of brown pigment, the *linea nigra,* below the umbilicus.

The *linea semilunaris,* upon either side of the abdomen, extends with a slight convexity outward from the junction of the tip of the ninth costal cartilage with the external border of the rectus muscle to the pubic spine. It corresponds accurately to the external limit of the sheath of the rectus muscle.

Lineæ transversæ are to be noted in subjects of good muscular development. They are transverse constrictions in the rectus muscles. They are three in number, as a rule, although a fourth may sometimes be encountered. One constriction is located at the level of the ensiform cartilage, another at the level of the umbilicus, and a third constriction midway between these two. When a fourth constriction is present, it is located midway between the umbilicus and the symphysis pubis.

Cutaneous flexion folds are encountered in obese subjects. They are usually two in number, one at the level of the umbilicus and the other just above the symphysis pubis.

TOPOGRAPHIC ANATOMY

The course of the *abdominal aorta* corresponds to a vertical line upon the anterior surface of the abdomen extending from a point a little to the left of the ensiform process downward to a point three-fourths of an inch below and a little to the left of the umbilicus, where the vessel bifurcates to form the common iliac arteries. Upon this line the celiac axis arises from the abdominal aorta at a point four and one-half to five inches above the umbilicus; the superior mesenteric artery arises at a point four inches above the umbilicus; the renal artery arises three and one-half inches above the umbilicus; and the inferior mesenteric artery arises one inch above the level of the umbilicus.

The course of the *common iliac* and *external iliac* arteries corresponds to a line drawn from the point of bifurcation of the abdominal aorta to a point midway between the anterior superior iliac spine and the symphysis pubis.

The course of the *deep epigastric* artery is represented by a line drawn from the midpoint of Poupart's ligament upward and inward to the umbilicus.

The course of the *inferior vena cava* is represented by a vertical line drawn along the line representing the course of the abdominal aorta, a little distance to the right side of this line.

The *common* and *external iliac* veins are indicated by lines upon the abdominal surface slightly below and to the right of and corresponding in direction to the lines of the arteries of the same name.

The topographic anatomy of the various abdominal organs is described in the sections dealing with the respective organs.

TOPOGRAPHIC LINES AND REGIONS

For purposes of description and in order to facilitate the accurate localization of pathologic conditions arising within the abdominal cavity, the abdomen is divided into certain arbitrary regions by means of vertical and horizontal lines, or the division of the abdominal cavity may be established by the utilization of the bony landmarks as detailed in a subsequent paragraph.

A very useful division of the abdominal cavity is by the erection of two vertical and two horizontal lines upon the anterior abdominal surface, thus dividing the abdominal cavity into nine regions. In this method of division of the abdominal cavity, the superior horizontal line, the *subcostal line*, is drawn around the body at the level of the most dependent portion of the tenth costal cartilage. The inferior horizontal line, the *intertubercular line,* encircles the trunk at the level of the tubercle which is palpable upon the iliac crest approximately two inches behind the anterior superior iliac spine. The two vertical lines, the *mid-Poupart lines,* are erected from the midpoint of Poupart's ligament upon either side, blending superiorly with the midclavicular lines of the thorax.

Through the medium of these arbitrary lines, the abdominal cavity is divided into nine regions. The *epigastric region*, bounded inferiorly by the subcostal line, and superiorly and laterally by the line of the costal arch, overlies the stomach, duodenum, liver, gall bladder, pancreas, and portions of the two kidneys.

The *left hypochondriac* region, limited inferiorly by the subcostal line and internally by the line of the left costal arch, overlies the fundus of the stomach, the spleen, and the splenic flexure of the colon.

The *right hypochondriac* region, limited inferiorly by the subcostal line and internally by the line of the right costal arch, overlies the portion of the abdominal cavity which is occupied by the liver and right kidney.

The *umbilical region*, limited superiorly by the subcostal line, inferiorly by the intertubercular line, and laterally by the right and left mid-Poupart lines, overlies the coils of the small intestine, the mesentery, the great omentum, a portion of the two kidneys, and of the transverse colon.

The *left lumbar region*, bounded superiorly by the subcostal line, inferiorly by the intertubercular line, and internally by the left mid-Poupart line, overlies the left kidney, the descending colon, and some coils of the small intestine.

The *right lumbar region*, limited superiorly by the subcostal line, inferiorly by the intertubercular line, and internally by the right mid-Poupart line, overlies a portion of the right kidney, of the ascending colon, and coils of the small intestine.

The *hypogastric region*, lying below the intertubercular line and limited laterally by the mid-Poupart lines, and inferiorly by the pubic symphysis, overlies the distended bladder, some coils of the small intestine, a portion of the sigmoid flexure, the cecum, occasionally the vermiform appendix, and the pregnant uterus.

The *left iliac region*, limited superiorly by the intertubercular line, inferiorly by Poupart's ligament, and internally by the left mid-Poupart line, overlies the sigmoid flexure of the colon.

The *right iliac region*, limited by the intertubercular line, the right mid-Poupart line, and Poupart's ligament, overlies the cecum and usually the vermiform appendix.

Instead of the division of the abdominal cavity into nine regions by means of two horizontal and two vertical lines, the cavity may be divided into four regions, or quadrants, through the medium of a horizontal and a vertical line passing through the umbilicus. In this method of subdivision the four regions are termed, respectively,

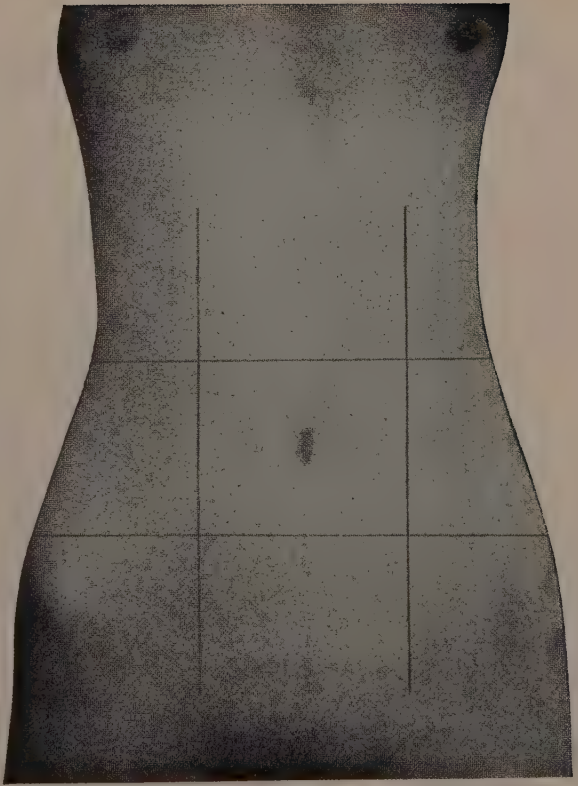


Fig. 20.—The usual anatomic division of the abdomen into nine regions by two transverse lines and two vertical lines. The upper transverse line is at the level of the cartilages of the tenth ribs, and the lower with the highest points of the iliac crests. The two parallel vertical lines pass through the cartilage of the eighth ribs and the middle of Poupart's ligaments. (From Crossen.)

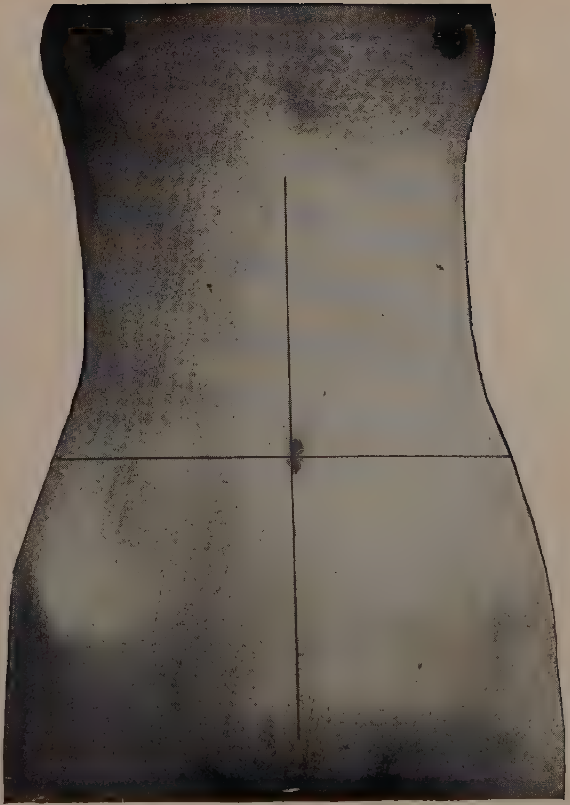


Fig. 21.—The abdominal surface divided into quadrants. (From Crossen.)

the right upper quadrant, the left upper quadrant, the right lower quadrant, and the left lower quadrant, of the abdomen.

The *right upper quadrant* of the abdomen overlies the right lobe of the liver with the gall bladder, the hepatic flexure and the proximal portion of the transverse colon, the pylorus, the first and second portions of the duodenum and the head of the pancreas, and more deeply in this region, the superior half of the right kidney.

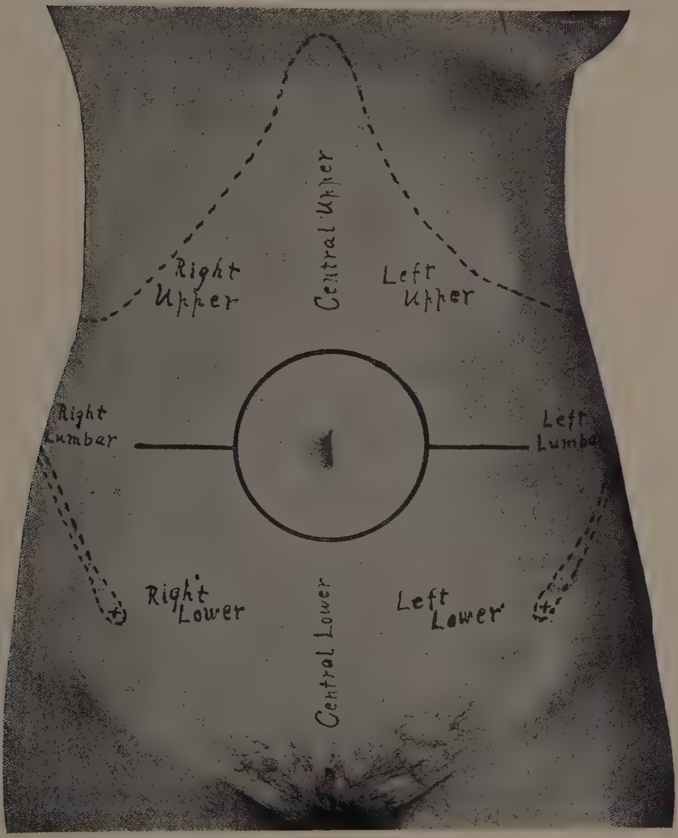


Fig. 22.—Another abdomen divided with the circle and short horizontal lines, and showing the names on the primary regions. The area within the circle carries the usual designation, "umbilical region." (From Crossen.)

The *left upper quadrant* of the abdomen overlies the left lobe of the liver, the stomach, the distal half of the transverse colon with the splenic flexure, the larger portion of the pancreas, the superior half of the left kidney, and the spleen.

The *right lower quadrant* of the abdomen overlies the cecum with the vermiform appendix, the ascending colon, the inferior half of the right kidney, the right fallopian tube and ovary, together with portions of the bladder and the uterus.

The *left lower quadrant* of the abdomen overlies the descending colon and sigmoid flexure, a portion of the inferior half of the left kidney, coils of the small intestine, the left fallopian tube and ovary, and a portion of the bladder and of the uterus.

A very convenient subdivision of the abdominal cavity is that devised by Crossen, in which the natural landmarks of the abdomen are utilized, the only artificial lines employed being one encircling the umbilicus, and a horizontal line drawn from either side of the circle. By this method the abdomen is subdivided into regions which are respectively designated as right upper, left upper, central upper, right lower, left lower, central lower, umbilical, right lumbar, and left lumbar.

SECTION II

PRELIMINARY OBSERVATION

Before starting a physical examination proper, certain observations should be made which do not properly belong under any of the subdivisions of the examination.

CHAPTER IV

SPEECH, GAIT, STATION

SPEECH

The speech should be observed while taking the history or during preliminary examination. A nasal quality of speech may result from nasal obstructions, such as occur with the common cold or hypertrophied adenoid tissue and paralysis or perforation of the soft or hard palate. Tongue-tie produces a peculiar lisping speech. Laryngitis, tumors or paralysis of the vocal cords, and disease or pressure on the recurrent laryngeal nerve, produces a hoarseness which may amount to almost complete aphonia.

Diseases of the central nervous system are often associated with peculiarities of speech, such as the scanning speech of multiple sclerosis.

While observing speech the general mentality and mental state may be considered. Markedly subnormal mentality may be determined by a few questions, while border-line cases require careful observation by a psychiatrist. Undue emotion, depressed or euphoric states, fear, inattention, memory, etc., should be noted.

THE GAIT

The gait should be observed both in slow and rapid locomotion, stopping, starting, and turning, as abnormalities may be present in all or only one or two movements. One should become familiar with the hesitating gait of the infant who is learning to walk, the springing step of the muscular adult, the faltering gait of the individual convalescing from wasting disease or overcome by weariness, and the uncertain gait of the aged. All of these may be normal under proper circumstances, while in another setting they may be distinctly pathologic.

In many nervous diseases the gait is characteristic and gives at once a clue to the correct diagnosis. In observing the gait of a patient who is suffering with an organic nervous disease the clothing should be removed from the lower extremities so that the phenomena attending locomotion may be clearly observed. Aids to locomotion, such as canes and crutches, should be noted and the exact reason for their use should be ascertained.

The Spastic Gait.—In spastic diplegia due to lesions in the lateral pyramidal tracts the lower limbs are stiff, owing to an inability to bend the knees, so that the patient progresses by means of short steps, the toes scraping along the floor. The toes of the shoes are worn excessively. The presence of a marked ankle clonus on both sides communicates a general tremulousness to the entire carriage of the patient, who is apt to stumble over slight obstacles and fall.

The Hemiplegic Gait.—The hemiplegic gait is merely a unilateral spastic gait, the spastic limb during progression describing an arc of a circle while the sound limb supports the weight of the body. In spastic cerebral paraplegia, or double hemiplegia, both limbs describe the arc of a circle during progression, each foot in turn being swung outward and planted in front of the other with the production of the cross-legged or "scissor" gait, the trunk and upper limbs meanwhile being jerked about from side to side in the effort to move the spastic members forward.

The Steppage Gait.—Patients with multiple neuritis with foot drop, or with lesions of the lumbosacral region of the spinal cord, exhibit the steppage gait, a mode of progression in which each foot is alternately raised high, the toe thrown upward, the foot striking the ground forcibly, as if the patient were continually stepping over obstacles in his path. A unilateral steppage gait accompanies paralysis of the external popliteal nerve.

The Ataxic Gait.—The ataxic or tabetic gait occurs typically in tabes dorsalis, a very similar gait being observed in Friedreich's ataxia, and in tumor of the posterior columns of the cord. The patient walks on a very broad base, swaying from side to side. The foot in progression is raised suddenly from the floor, is thrown forcibly forward, and thrown forcibly down "in flail-like fashion," the heel usually striking the floor first. The patient keeps the eyes fixed steadily upon the floor before him in the effort to guide his onward course. He is unable to suddenly stop or start on command or to turn suddenly and retrace his course. Similar ataxia in the upper limbs is demonstrable in the inability of the patient to touch the finger tip to the nose, or to accurately approximate the finger tips with the arms before the body.

The Festinating Gait.—In paralysis agitans the patient moves forward with the body inclined somewhat forward, advancing with short, shuffling steps which become progressively faster as he crosses the room. When ordered to turn, the entire body is turned *en masse.* This type of locomotion constitutes the so-called *propulsion. Retropulsion* may often be elicited in these patients. If the patient is quickly pulled backward, and, indeed sometimes on merely looking upward, he tends to run backward with short, shuffling steps, although the body is invariably inclined forward.

The Cerebellar or Vertiginous Gait.—In cerebellar disease associated with severe vertigo the patient progresses in a very irregular course, often lurching from side to side. Quite frequently the patient exhibits a tendency to reel in a fixed direction, forward, backward, or to one side. This type of progression occurs with unilateral cerebellar lesions. In unilateral cerebellar tumors the head is not infrequently inclined toward opposite side, while the face is turned slightly toward the side of the lesion.

THE STATION

The station is the attitude of the patient when standing at ease in the erect posture. In testing the station the patient should be directed to stand with the feet closely approximated, and the test should be made first with the eyes open and then with the eyes closed. A normal person while undergoing this examination will frequently sway slightly from side to side, and in cases of muscular weakness, either from exhausting disease or from neurasthenia, the swaying is more marked. But when the swaying movement becomes so extreme that the patient is in danger of falling if not supported, the station becomes pathologic. Thus, in tabes dorsalis the patient with feet closely approximated and the eyes closed sways excessively; and if not supported, is apt to fall (Romberg's sign).

CHAPTER V

HABITUS OR BUILD

Individuals may be roughly divided into three groups, asthenic, sthenic, and hypersthenic.

The asthenic individual is characterized by a long neck, narrow shoulders and thorax, with a narrow epigastric angle, and sometimes floating tenth ribs. The abdomen is often more prominent in the lower third. The musculature is likely to be poorly developed.

The sthenic type presents a moderately short neck, fairly wide shoulders, and a fairly well developed chest, with epigastric angle equal to about a right angle. The abdominal wall is muscular and is more prominent just below the umbilicus. The musculature is of good development.

The hypersthenic type has a short neck, usually with considerable cutaneous fat. The thorax is short, with a wide epigastric angle, and the abdomen is prominent with an excess of subcutaneous fat.

The asthenic type is more prone to pulmonary tuberculosis, ptosis of the stomach and abdominal organs, and is often possessed of a more highly sensitive sensory nervous mechanism, rendering him more susceptible to functional nervous disorders.

The sthenic type is usually more evenly balanced both physically and nervously, and when ill will be found to be suffering from infections or accidents more frequently than from functional disorders.

The hypersthenic type tends to become obese. The heart is usually transverse and tends to hypertrophy from the constant strain associated with carrying excess of fat. Arterial hypertension and degenerative diseases of the heart, arteries and kidneys are more common in this type.

Naturally, all graduations between asthenic and hypersthenic occur. Habitus is frequently a family or racial characteristic, but variations from a family type are extremely common.

HEIGHT

Height should be studied especially with reference to age and weight. The average individual stops growing taller at about the eighteenth year, and growth after the twenty-fifth year should excite suspicion of disease.

Diseases of the pituitary gland are associated with increase in height, due to increase in the length of the long bones. Practically all giants have pituitary abnormalities.

Height should be taken without shoes, the patient standing erect, as against a wall, with feet together. A bar across the top of the head should be at right angles to the wall or measuring bar.

WEIGHT

The weight of an individual is so influenced by surroundings, diet, exercise, climate, etc., that before ascribing changes in weight to disease, a careful analysis of other factors should be made. To weigh a patient correctly, clothing should be removed, or subsequently weighed. All future weights should be recorded under approximately the same conditions, as food, drink, exercise, urination and defecation influence weight materially. A glass of water adds 6 to 8 ounces, and a couple of sets of tennis may cause the loss of two pounds through excessive perspiration.

Children gain weight rapidly during the first years of life; usually the weight doubles in the first year and again doubles by the third year. Thereafter, the gain is slow, but steady, until the maximum height is reached, about the eighteenth year.

Boys and girls vary somewhat in weight. Girls often outweigh boys of the same age and height up to puberty, after which the average weight of the boy is greater than the girl. During adult life weight, subject to environment and disease, runs constant, to increase in the female after the menopause and the male after forty.

The degenerative changes of old age are usually associated with loss of weight. Seasonal changes of weight in the adult are worthy of note. In temperate climates the average adult gains weight from September to January, remains constant from January to April or May, and gradually loses during the summer months. This seasonal variation is from four to twenty pounds. The recognition of seasonal changes is of value in the study of disease, as false assurance may develop if a patient remains constant or gains slightly during the fall, while one may be unduly concerned over a loss occurring in the summer months. Change from a tropical to a temperate or cold climate is usually associated with gain in weight. Change of occupation may cause either gain or loss of weight, the gain being especially noticeable in the muscular individual who changes from active to sedentary occupation. The loss of weight is especially noticeable in change from day to night work and from sedentary to active occupations. Gain in weight during early pregnancy is often more than the weight of the uterine contents would account for. Patients after recovery from a wasting disease, such as typhoid, often gain in weight for many months.

Gain in weight does not always speak for well-being, as the obesity of patients with disturbed thyroid and pituitary functions will attest. Collections of fluid in the subcutaneous tissues or serous cavities may cause gain in weight, while actual loss of fat and muscle occurs. Newgrowths may also cause actual gain in weight, with loss of fat and muscle. This is particularly true of ovarian cysts. Many insane persons gain rapidly in weight.

Loss of weight on the other hand does not necessarily mean disease. The converse of the seasonal gain above mentioned is the seasonal loss. Removal of carbohydrates and fats from the diet, so popular at present, should be investigated before ascribing loss of weight to disease. Occupational changes and loss of sleep, together with changes from temperate to tropical climates, should not be forgotten.

Pathological loss of weight may result from various types of disease. Insufficient or unbalanced diet is accountable for loss of weight in adults, as well as in infants. The inability to take or retain foods, as in esophageal stenosis, and vomiting and diarrhea cause severe drain on the fat reserve.

Due to functional and organic diseases of the digestive tract, food may be improperly prepared for absorption, so that only a small amount of the food taken is metabolized. Infectious diseases, such as typhoid fever, pneumonia and tuberculosis may increase oxidation, thus producing undue waste or the toxins produced may influence digestion and absorption.

Malignant disease produces emaciation by obstructing intake, increasing elimination, or influencing metabolism. Mental derangements by reducing sleep and rendering patients afraid to take food often cause marked loss of weight.

CHAPTER VI

BODY TEMPERATURE

The temperature of the body is determined by the aid of a clinical thermometer. This thermometer differs from the ordinary thermometer in that it has a shorter tube with a reading scale ranging from 34 to 42° C. or 94 to 110° F. Just above the mercury bulb is a constriction in the lumen of the tube. This constriction inhibits the return of the mercury as the temperature of the bulb falls, thus maintaining the column at its highest point until it can be observed, and renders it necessary to "shake down" the mercury column before attempting another reading. The mercury tube is also constructed with a lense which magnifies the mercury column sufficiently to facilitate reading.

Unfortunately, in the United States both the Fahrenheit and centigrade scales are in use, so that the following formula must constantly be kept in mind:

Centigrade temperature $=$ Fahrenheit temperature $- 32 \times 5 \div 9$.
Fahrenheit temperature $=$ Centigrade temperature $\times 9 \div 5 + 32$.

Temperature may be taken by placing the thermometer under the tongue and closing the lips (sublingual), by inserting the thermometer about one and one-half inches into the rectum (rectal), or by placing it in the axilla and holding the arm firmly against the side (axillary). The rectal temperature is the most accurate, but the sublingual is more convenient and is sufficiently accurate for clinical work. The axillary temperature is less accurate and should not be used when the other methods are available. Using the sublingual method as a standard, the rectal temperature is considered about six-tenths of a degree higher and the axillary temperature one degree lower. To secure accurate readings, a thermometer should be left in place at least three minutes.

A body temperature between 36° to 37° C. (98° to 98.8° F.) is considered normal. Below this, subnormal, and above, fever. (There are a few individuals without demonstrable disease who show a constant temperature above or below normal.) Temperature varies slightly during the twenty-four hours, being lowest in the early morning and highest at the close of the working period (about 5 or 6 P.M.). There is also a slight elevation after a heavy meal or after excessive exercise.

Subnormal temperatures occur in heart disease when decompensation exists, in degenerative kidney lesions, in wasting diseases, such as cancer, and in heat stroke. Fever is the result of excessive oxidation due to infectious diseases, certain intoxications, and sun stroke.

SECTION III

INSPECTION

Inspection, while the simplest procedure in physical diagnosis, requiring only a careful scrutiny of the region under consideration, is often the most neglected. In the following pages an attempt is made to outline a technic for systematic inspection. References to pathologic conditions of the various organs is made essentially to impress the observer with the importance of the observation under discussion and not as a catalogue of all the ills to which the organ is susceptible.

CHAPTER VII

INSPECTION OF THE HEAD, FOREHEAD, AND EARS

THE HEAD

When inspecting the head the following points should be noted by the examiner:

1. Size and shape.
2. Condition of the fontanelles and sutures (in children).
3. Condition of the bones and scars.
4. Condition of the hair.
5. Position of the head.
6. Movements of the head.

Size and Shape.—The head may be abnormally small (microcephalia) with premature closure of the fontanelles and sutures, a condition usually associated with idiocy.

A very large head is encountered in hydrocephalus, while moderate enlargement occurs in connection with rickets, cretinism and hypertrophia cerebri.

Hydrocephalic Head.—In hydrocephalus the head is enlarged, the circumference sometimes reaching 32 inches at the eighth month of life. The large prominent forehead is in marked contrast with the small face. The fontanelles and sutures are widely open, and the veins of the scalp are prominent and distended.

The skull is very thin and may be translucent to candle light. The child has difficulty in holding the head up.

In *rickets* the circumference of the head is increased two or three inches, the enlargement being chiefly due to thickening of the cranial bones. The shape of the head is rather square (box head), owing to the presence of osteoid bosses upon the frontal and parietal regions. The rachitic head is flattened at the vertex and over the occiput. Soft, compressible areas, *craneotabes,* are often present; the fontanelles are widely open; and the sutures are tardy in closing.

While the head in hydrocephalus somewhat resembles the rachitic head, there are differences. In hydrocephalus the shape is globular rather than square as in rickets; also in hydrocephalus the sutures and fontanelles are wider and the fontanelles bulge, which is not true of rickets.

In *cretinism* the head is large, flattened at the vertex, with open sutures and fontanelles, but without bulging.

Fontanelles and Sutures.—The posterior fontanelle normally closes about the end of the second month, while the anterior fontanelle closes between the eighteenth and twentieth months of life.

Tardy closure of the fontanelles occurs most frequently in connection with rickets. Closure is also delayed in hydrocephalus and cretinism. In rickets the fontanelles may remain open beyond the fourth year of life.

Bulging fontanelles indicate increased intracranial pressure, and are noted in hydrocephalus, cerebral hemorrhage, meningitis, brain tumor, sinus thrombosis, meningeal hemorrhage, and during acute fevers.

Depressed fontanelles are noted during chronic wasting diseases, in pulmonary diseases attended by dyspnea, after severe diarrhea, during the early stages of meningitis, and in cholera infantum.

Enlargement of the fontanelles, the anterior fontanelle exceeding one inch in diameter, is suggestive of rickets, hydrocephalus, or cretinism, but may be a hereditary condition.

Open Sutures.—The sutures of the child's head normally close between the sixth and eighth months. Open sutures after this time are significant of rickets, cretinism, or hydrocephalus.

Condition of the Bones of the Head.—A number of changes in the bones of the head possess diagnostic significance.

Osteoid bosses on the frontal and parietal bones in infants are symptomatic of rickets.

Soft, nodular swellings on the skull, which become harder with advancing age, are symptomatic of syphilitic periostitis of the cranial bones.

Scars on the head, whether from accidents or from surgical trauma, should be noted and checked by palpation.

Tumors of the scalp may be due to trauma, new growths, or sebaceous cysts.

The Condition of the Hair.—*General falling* of the hair follows many acute febrile conditions, notably typhoid fever. General loss of hair also occurs in gout and myxedema.

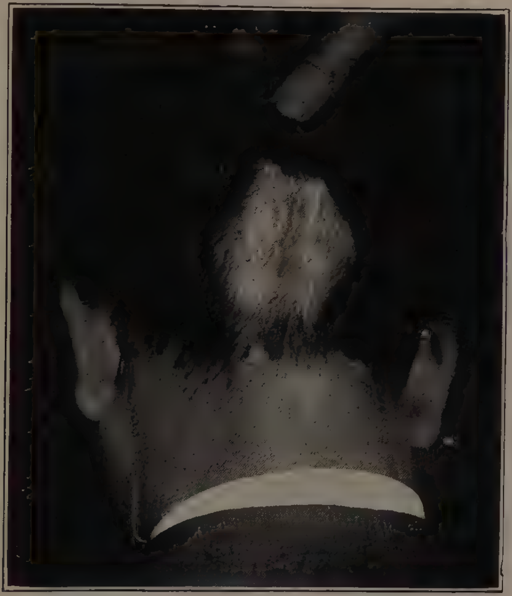


Fig. 23.—Alopecia areata. A patch that is not as yet completely denuded. (From Hazen.)

Circumscribed falling of the hair, producing local areas of baldness, results from tinea tonsurans, scarring from local trauma, neuralgia of the trigeminal nerve, and syphilis.

In a child baldness in the occipital region, with excessive sweating of the head suggests rickets.

Baldness in early middle life is often a family characteristic.

Color of the Hair.—The color of the hair may be altered by local application of chemicals, or as a result of metallic poisoning. Thus, hydrogen dioxide bleaches the hair, while the hair assumes a green color in chronic copper poisoning.

Canities, whiteness of the hair, a physiologic change in persons past middle age, is observed in connection with syphilitic endoarteritis involving the scalp and accompanying trophic nervous disturbances. Premature whitening of the hair is occasionally seen in otherwise normal people.

Position of the Head.—*Retraction* of the head occurs in tetanus, strychnine poisoning, and meningitis. In children retraction of the head may occur during attacks of acute indigestion, the significance of which is slight; but it should not be mistaken for retraction in connection with grave affections.

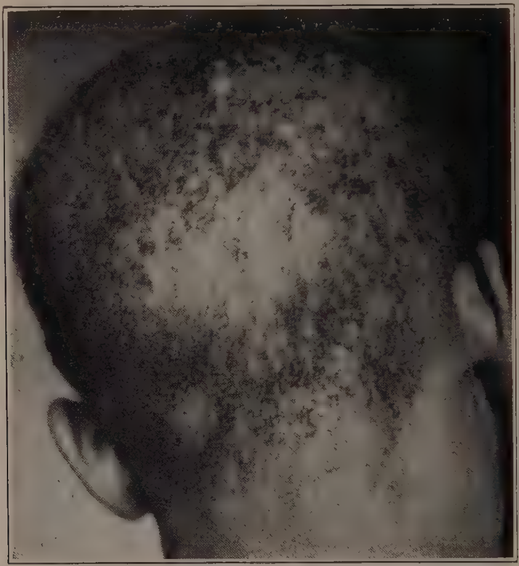


Fig. 24.—Syphilitic alopecia. (From Hazen.)

Lateral deviation of the head is observed in connection with *wry-neck* due to spasm of the sternomastoid muscle, in rheumatic torticollis, in which there is painful contraction of the sternomastoid and trapezius, and in hematoma of the sternomastoid, which is attended by an oval tumor in the belly of the muscle. In young children lateral deviation of the head may be due to injury to the muscles of the neck during parturition.

Abnormal fixation of the head is observed in connection with retropharyngeal abscess, cervical adenitis, rheumatism, arthritis deformans, extensive scars from burns, and in tuberculosis of the cervical spine.

Movements of the Head.—*Nodding spasm,* a rhythmical up and down movement of the head, is observed in patients suffering with hysteria, and occasionally in connection with rickets. The movement may be continuous; or may be absent during quiescence and only brought out by excitement.

Arrhythmic, purposeless movements of the head occur in Sydenham's chorea.

Spasmodic movements of the head, in which the head deviates laterally, occur in spasmodic torticollis.

Inability to move the head occurs in connection with the flaccid paralysis of acute anterior poliomyelitis, in caries of the cervical vertebræ, in the late stages of cerebrospinal meningitis, and during comatose states from any cause.

THE FOREHEAD

The forehead should be examined for scars, skin eruptions, and nodular swellings.

Scars upon the forehead may be indicative of former traumatism or of the eruption of syphilis.

Eruptions.—The forehead is subject to many cutaneous eruptions, notably those of measles, smallpox, syphilis, and acne. All eruptions should be carefully studied.

Nodular swellings of the forehead may be indicative of glanders, trichinosis, syphilitic periostitis, or tumor of the cranial bones.

THE EARS

In routine physical examination the external ears should be carefully examined as to size, shape, and color. Comparison of the ears is essential.

Congenital Defects.—Among the congenital defects of the auricle may be mentioned entire absence of this portion of the auditory apparatus; excessive development or defective development of the auricle, macrotia and microtia respectively; the presence of more than one auricle, or polyotia; and malformation or absence of the lobule, helix, or antihelix.

Fistula auris congenita, a rare defect, consists of a short blind canal, lined with epithelium, with its orifice either in front of or below the tragus.

Hematoma auris, or *othematoma,* is a bluish-red swelling involving the concha and fossa of the antihelix and helix, the lobule escaping. It is a trophoneurosis, and the condition is observed most commonly among insane patients, in whom it was formerly attributed to ill-treatment. A similar bluish discoloration, involving the entire auricle may follow trauma, due to effusion of blood beneath the perichondrium.

Blueness of the auricle occurs as a sign of cyanosis, and also in the early stages of frostbite, in which it becomes later yellowish-white.

Redness of the auricle follows slight irritation and is commonly seen in individuals who blush under certain types of mental excitation. It also occurs with vasomotor syphilis.

Tophi, small, hard nodules of sodium urate, are frequently found in the helix in gouty patients.

Cysts of the auricle, small, noninflammatory tumors, containing clear fluid, are sometimes encountered about the auricle. They are differentiated from perichondritis by the absence of pain and other inflammatory signs.

Sebaceous cysts, due to blocking of the ducts of the sebaceous glands and accumulation of the secretion, produce round tumors situated usually in the skin behind the lobule or in the lobule.

Keloid may be encountered on the lobule, due to piercing the lobule for earrings, most commonly in the negro race.

Otomycosis.—In otomycosis, due to the growth of the aspergillus niger in the external auditory canal, the canal is studded with black spots, which under the microscope reveal the presence of the fungus.

Discharge of blood from the external auditory meatus is indicative of fracture of the base of the skull or otitis media. In the case of fracture of the base of the skull the blood is mixed with cerebrospinal fluid, which prevents coagulation; while in otitis media there is admixture with pus. Discharge of pus unmixed with blood indicates purulent otitis media or abscess.

Inspection of the ear drum and auditory canal requires the use of an ear speculum and reflected light. The normal ear drum is grayish white with an indentation just below the center corresponding to the attachment of the malleus. The reflected light thrown on the membrane shows a lighter rod-shaped marking known as the "cone of light." A careful examination of the ear drum should be conducted in an ear clinic and possible abnormalities discussed by an otologist.

The mastoid regions should be inspected and compared. Redness and swelling of the mastoid region usually indicates inflammatory disease of the mastoid cells, but may occur in erysipelas.

CHAPTER VIII

INSPECTION OF THE FACE, THE EYES, AND THE NOSE

THE FACE

Contour of the Face

Observation of the contour of the face and facial expression should be cultivated so that in our ordinary contacts such a study is almost subconscious.

The contour of the face is altered by many diseases, chief among which may be mentioned acromegaly, hydrocephalus, osteitis deformans, leontiasis ossium, leprosy, and facial hemiatrophy and hemihypertrophy.

Acromegaly.—In acromegaly the face assumes an oval or elliptical shape, due to the enlargement of the frontal and malar bones, and the mandible, which become massive. Owing to this growth the teeth are separated by intervals, the lower teeth projecting beyond those of the upper jaw. The ears are large, the nose thickened, and the superciliary ridges are prominent. The tongue is large, sometimes protruding from the mouth. The eyes are unchanged; and, by contrast with the massive features, appear abnormally small.

Cretinism.—In cretinism the face is broad and flat, presenting a bloated appearance. The eyes are wide apart, the eyelids are thickened, and the nose is broad and negroid. There is pouting of the lips and protruding tongue, the child presenting a picture of imbecility.

Myxedema.—In myxedema the lines of expression in the face are obliterated by swelling in the subcutaneous tissue. The contour of the face has been likened to a "full moon." The nostrils and lips are large and thick, the mouth is enlarged, and there is usually a reddish patch over the cheek.

Hydrocephalus.—The face in this disease is triangular with the base of the triangle above. The features, which are of normal size, present a marked contrast with the enormous forehead.

Osteitis Deformans.—The face in this disease is triangular with the base directed upward, owing to the thickening of the

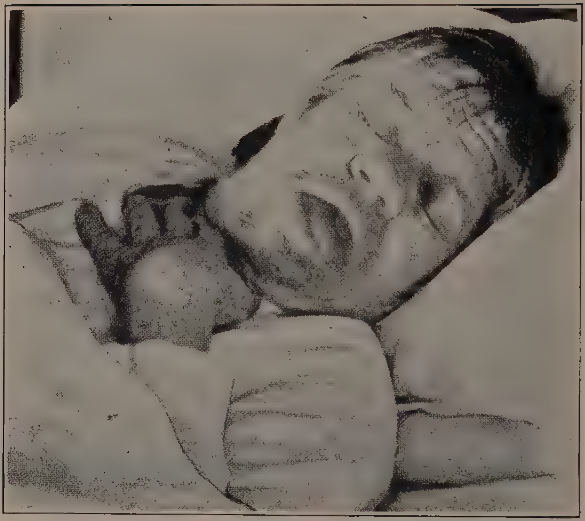


Fig. 25.—Face of acromegaly. (Butler, after Worchester.,

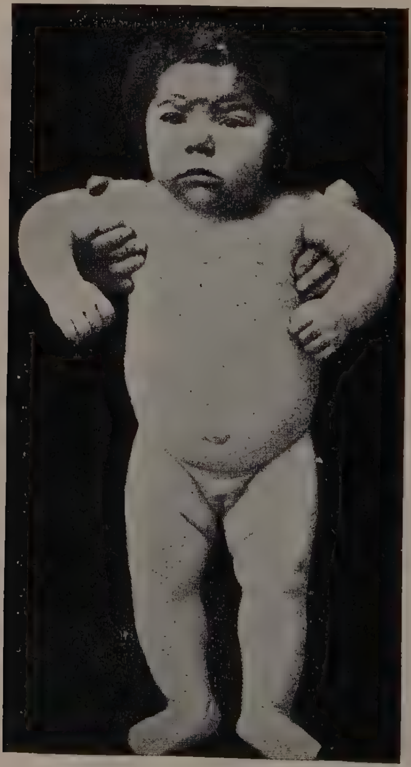


Fig. 26.—A case of congenital myxedema. (Woolley, after Kassowitz.)

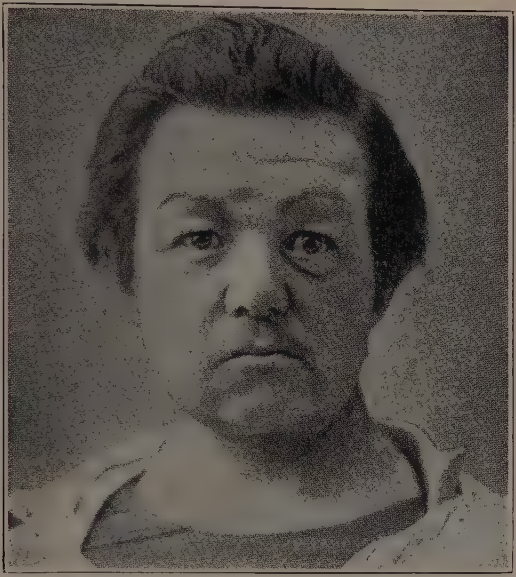


Fig. 27.—Face of myxedema. (Butler, after Gardinier.)

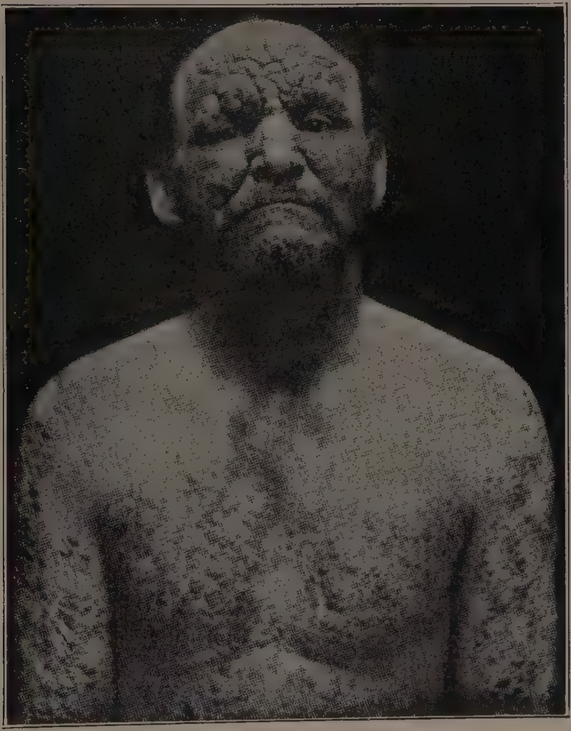


Fig. 28.—Leprosy. (From McFarland.)

bones of the cranium. The head is carried in a position of forward inclination.

Leontiasis Ossium.—This disease is characterized by progressive enlargement of the bones of the cranium and face, beginning usually in the superior maxillary bones. Blindness occasionally develops from pressure upon the optic nerves.

Leprosy.—When the nodes of leprosy develop in the face they produce thickening of the skin of the forehead and cheeks. The nose is flat and thick; the lips are thick; the ears are thick and large, while the eyebrows, eyelashes, and beard are shed, constituting the facies leontina.

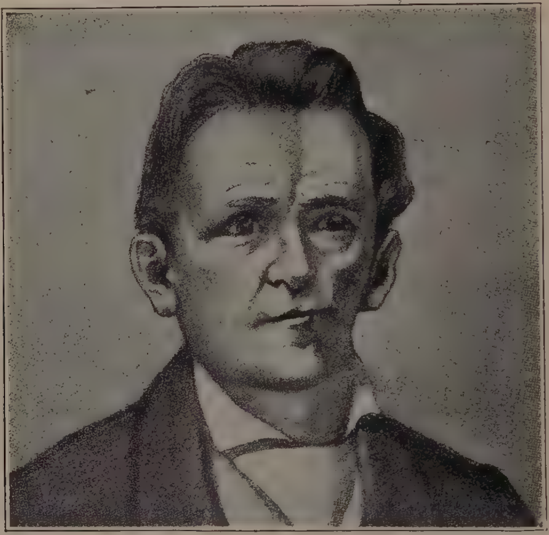


Fig. 29.—Facial hemiatrophy. (From Butler.)

Facial Hemiatrophy.—In facial hemiatrophy one-half of the face is smaller than the opposite half, with a sharply defined vertical line of junction. The condition usually begins during childhood in one or two spots on one side of the face. The skin begins to undergo atrophic changes, followed by a similar involvement of the underlying subcutaneous tissue, muscles and bones. The skin of the affected half of the face becomes wrinkled, the teeth become loose, and the eyebrows fall out. The secretion of the sebaceous glands is diminished or abolished. The face is drawn toward the sound side, rendering the contrast between the two sides striking.

A similar facial asymmetry is encountered in children as a developmental defect, often in association with congenital torticollis.

Facial hemihypertrophy, the opposite condition, in which one side of the face is enlarged, occurs as an anomaly in the development of the face, sometimes associated with hemihypertrophy of the entire half of the body.

Color of the Face

The normal color of the face varies with race and complexion. The yellowish tint of the oriental, the brown to black color of the Negro, and the coppery tint of the American Indian are matters of common knowledge. Racial mixtures give rise to various modifications of color. The blonde and brunette types, with their variations, should also be borne in mind.

Pallor of the face occurs in anemia, ischemia, the edema of Bright's disease, and transiently as the result of sudden fright.

Flushing of the face may be transient and due to vasomotor disturbance, or may be persistent, notably in the early stage of acute fevers, such as yellow fever, pneumonia, and tuberculosis. A flushed face accompanies excessive cardiac hypertrophy, and in one form of essential anemia, namely, chlorosis rubra, is a marked feature of the disease. In apoplectic attacks and in the early stages of alcoholic intoxication the face is flushed.

Cyanosis, or bluish discoloration, particularly noticeable in the lips and ears, occurs in uncompensated heart disease. A similar bluish discoloration of the face is symptomatic of poisoning with coal tar products.

Yellowish discoloration of the entire face is suggestive of the cachexia of malignant disease, syphilis, or chronic malaria. A similar hue accompanies chronic constipation with inactive liver, certain cases of exophthalmic goiter and Addison's disease. A lemon yellow color of the face and body, with maintenance of the subcutaneous fat of the body, occurs with pernicious anemia. Shades of yellow varying from a faint tinge to a deep copper color occur with obstruction of the bile ducts or malignant disease of the gall bladder.

Brownish, muddy patches upon the face, termed *chloasma,* frequently develop in pregnant women and in women with uterine or ovarian disease.

Spasm of the Face

Spasm of the facial muscles occurs as a result of functional or organic disorders. It may be tonic or clonic, unilateral, or bilateral. It is more frequently encountered in women than in men. Among the conditions in which facial spasm possesses diagnostic significance may be mentioned:

Habit Spasm.—This spasm occurs in neurotic children, particularly in young girls. It is intensified by excitement or examination. It may consist in the rapid winking of an eye or the drawing up of one corner of the mouth. The neck muscles are frequently involved, the head being given a quick shake at the time of the winking.

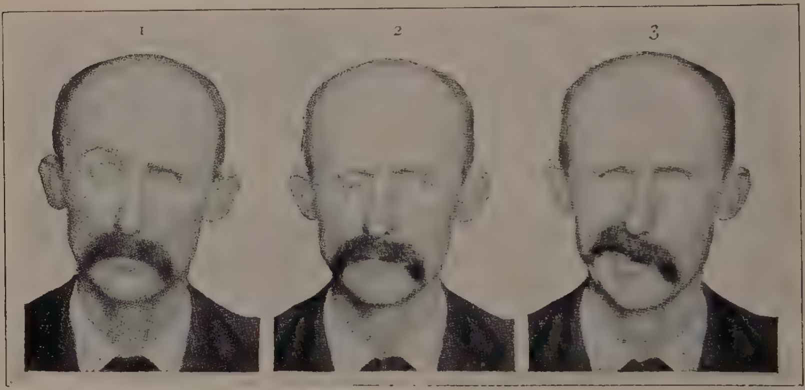


Fig. 30.—Facial paralysis. (Church.) *1*, bilateral attempt to raise eyebrows; *2*, bilateral attempt to close eyes; *3*, smiling. (From Eisendrath.)

Convulsive Tic.—This is a very sudden spasm of the facial muscles, frequently involving the brachial muscles as well. The spasmodic movements may be almost constant or may occur in paroxysms. In extreme cases the spasm may involve all the muscles of the body, the movements being very irregular and violent. The spasm is often accompanied by explosive utterances, echolalia and coprolalia.

Blepharospasm.—This is a sudden tonic contraction of the orbicularis palpebrarum muscle, causing partial or complete closure of the eye. More commonly the spasm affects the lateral facial muscles also, producing constant twitching of the side of the face. Usually unilateral, blepharospasm may be bilateral. The spasm is increased by emotional excitement and voluntary movement of the muscles of the face. If not reflex from irritation of the conjunctiva or cornea by a foreign body, it indicates involvement of the facial nerve.

Chorea.—Chorea produces arrhythmical jerking contractions of the facial muscles. It is accompanied by the other symptoms of the disease, as purposeless movements of the hands and feet.

Exophthalmic Goiter.—Spasm of the levator palpebræ superioris muscle, causing rapid movements of the upper lids, occurs occasionally in exophthalmic goiter, in which it constitutes Abadie's sign of this disease.

Tetanus.—Tetanus or lockjaw produces tonic spasm of the facial muscles, with the risus sardonicus, or sardonic smile, which is characteristic of the disease.

Unilateral clonic spasm of one or more facial muscles points to irritation of the facial region of the cortex of the brain or to irritation of the facial nerve trunk in its course or at its exit by tumor or aneurysm of the vertebral artery.

Facial Paralysis.—In paralysis due to lesions of the seventh nerve the normal flexion folds disappear from the affected side of the face, the patient is unable to close the eye, which remains open and staring, is unable to whistle or smile, and the angle of the mouth droops on the paralyzed side, while the opposite angle is drawn toward the healthy side. These changes constitute the typical Bell's palsy, which is due to a lesion of the nerve after its exit from the stylomastoid foramen, and which is often due to exposure to cold.

Facial Expression

The facial expressions are difficult to describe, but expressions of pleasure, fear, anger, pain, grief, stupor, and lethargy are worthy of study in the normal, as occurring without the proper stimuli. They may be evidence of disease. Exophthalmic goiter is often associated with an expression of fear, cretinism with a lethargic expression, and abnormal mental strains with facial expressions out of keeping or out of proportion to the exciting cause.

THE EYES

The Eyelid

The lids should be examined for edema, color, movements, swelling, condition of the lashes, and tumors.

Edema.—Edema of the lids with puffiness, occurs in connection with the edema of nephritis and in anasarca due to cardiac disease or hepatic cirrhosis. Edema of the lids is also noticed during the active stage of pertussis, severe coryza, erysipelas, cerebral thrombosis, and in arsenic and iodine poisoning. Slight puffiness and edema of the lids upon arising in the morning is noted in certain persons as a normal phenomenon.

Redness of the lids occurs with slight irritation, as following weeping, infections, such as common colds, "pink eye," scarlet fever, and measles.

Duskiness of the lids and the infraorbital region is symptomatic of uterine and ovarian disease, pregnancy, anemia and exhausting disease, the molimina of menstruation, and it is said, masturbation. Not infrequently normal individuals exhibit this phenomenon.

Ptosis.—Ptosis of the upper lid is usually due to syphilitic paralysis of the oculomotor nerve. While usually unilateral, ptosis may be bilateral. A bilateral ptosis of brief duration sometimes is seen in anemic and overworked women. Ptosis of the lid also occurs with acute encephalitis and as a congenital condition.

Lagophthalmos.—Lagophthalmos, imperfect closure of the eyelids, occurs with the exophthalmos of Graves' disease, and as a result of partial seventh nerve paralysis.

Absence of the lashes follows acute infections and syphilis.

Hordeolum.—A hordeolum or stye is a small abscess in the lid margin, situated at the root of an eyelash upon the anterior margin of the lid. Styes are acute and run a short course, but are prone to recur repeatedly.

Blepharitis Marginalis.—Blepharitis marginalis, inflammation of the lid margin is characterized by the formation of a series of scales or crusts along the lid margin, which upon removal expose a red, glazed surface. As the scales adhere to the lashes, they are sometimes mistaken for the eggs of pediculi.

Chalazion.—A chalazion or meibomian cyst is a small hard tumor of the upper eyelid, imbedded in the tarsal plate. It results from obstruction of a meibomian gland, and is prone to become inflamed and suppurate.

Epithelioma.—Epithelioma usually is seen upon the lower lid in persons past middle life. A history of long duration is usually obtainable.

Spasm and tic are noted under discussion of the face.

The Conjunctiva

The conjunctiva should be examined as to color, swelling, and tears.

Pallor.—Pallor of the conjunctiva is a sign of anemia, and calls for rather than replaces a blood examination.

Yellowness of the conjunctiva accompanies jaundice, and points to hepatic disorder.

Subconjunctival hemorrhage may occur during paroxysms of cough in pertussis or asthma, or as the result of local trauma.

Conjunctivitis.—In inflammation of the conjunctiva the membrane is red and bathed with mucopurulent or purulent discharge. Conjunctivitis may result from local infection, or may accompany the acute infectious diseases.

Absence of tear secretion produces dryness of the conjunctiva which predisposes to ulceration.

The Globe

Each globe should be compared with its fellow as to size, prominence, and movements by external muscles, as tested by instructing the subject to follow an object to the right, to the left, upward, downward, and in a circle.

Position of the Globe.—During epileptic seizures and hysterical coma the globes of the eyes rotate and turn upward. In hydrocephalus the globe looks downward, while following cranial injuries both globes look toward the side of the injury (conjugate deviation).

Exophthalmos.—Exophthalmos, or protrusion of the globe of the eye, may be indicative of hemorrhage into the orbit, paralysis of the ocular muscles, thrombosis of the superior longitudinal sinus, tumor of the orbit or superior maxillary bone pushing the globe forward, or of exophthalmic goiter. Exophthalmos may be unilateral or bilateral, the latter constituting one of the cardinal symptoms of exophthalmic goiter.

Enophthalmos.—Recession of the globe of the eye into the orbit, enophthalmos, occurs in exhausting diseases, particularly those which are associated with the loss of tissue fluids, as cholera. Enophthalmos is also caused by absorption of the orbital adipose tissue during chronic wasting disease, notably in tuberculosis, diabetes, marasmus, and the cachexia of malignant disease. Recession of the globe often follows injury to the cervical sympathetic nerves or surgical sympathectomy. It is known as Horner's syndrome.

Von Graefe's sign of exophthalmic goiter consists in the inability of the upper lid margin to accurately follow the sclerocorneal junction downward during downward rotation of the globe of the eye.

Strabismus (Squint).—In paralysis of one or more ocular muscles the normal axis of the eyeball deviates from its normal position, with the production of double vision or diplopia. A simple rule in the differentiation of the various ocular paralyses is that the affected eye is displaced by the unopposed antagonists to the side opposite to the usual traction of the paralyzed muscle, while the false image, the result of diplopia, is displaced in the direction of the line of traction of the paralyzed muscle (Purves Stewart).

Nystagmus.—Nystagmus is a rapid oscillation of the globe of the eye upon voluntary motion, usually in a horizontal direction, more rarely in a vertical direction, and very rarely it is rotary. It is a sign of value in disseminated sclerosis, epilepsy, brain tumor, tabes dorsalis, Friedreich's ataxia, and in some cases of chorea. Nystagmus may be the result of errors of refraction and may be noted in albinos. Miners are subject to a form of nystagmus, probably caused by the constant excursion of the eyes while working in the recumbent or stooping posture.

Conjugate Deviation.—This comprises a concomitant deviation of both eyes toward the right or left, its significance being a lesion in the cerebral cortex, corona radiata, or internal capsule, above the crossing of the motor fibers. Thus, in cerebral hemorrhage the eyes are turned toward the side of the lesion and opposite to the side of the paralysis (Prevost's sign). In interpreting the sign it is to be remembered that the lateral movements of the eyes are governed by impulses arising in the cerebral cortex and passing by way of the corona radiata and internal capsule to the sixth nerve nucleus of the corresponding side, and thence across the posterior longitudinal fasciculus to the subdivision of the opposite oculomotor nerve nucleus which presides over the internal rectus muscle. Thus the conjugate lateral deviation of the eyes is caused by the simultaneous stimulation of the external rectus muscle on the side of the lesion and of the internal rectus muscle on the side opposite to the lesion, causing the patient to "look at his lesion."

Cornea and Sclera

The cornea and sclera should be examined for color and evidence of any deviation from the normal glistening surface. This is especially true of the cornea where opacities may lead to serious defects of vision.

Arcus Senilis.—The arcus senilis is a grayish line at the sclerocorneal junction which partially encircles the cornea. Present in many elderly persons the arcus senilis is particularly frequent in arteriosclerosis and chronic nephritis.

Interstitial Keratitis.—Inflammation of the interstitial tissue of the cornea, leading to partial opacity of this structure or to small pinkish ''salmon patches'' is nearly always a sign of hereditary syphilis. The condition is usually bilateral, affecting children between 5 and 15 years of age, girls being more frequently attacked than boys.

Ulceration.—Comparatively large ulcers of the cornea are apparent as losses of the surface epithelium; while minute ulcers may require the instillation into the eye of a few drops of Fluorescin, which stains the ulcer a bright yellow-green. Corneal ulceration frequently develops during the exposure of the cornea as a result of the exophthalmos of Graves' disease, and in cases in which the cornea is insensitive owing to disease of the ophthalmic division of the fifth cranial nerve. Corneal ulcer is prone to develop during prolonged fevers when the patient lies long with the eyes only partially closed.

Opacity.—Corneal opacity may result from the repair of a corneal ulcer, from interstitial keratitis, or as a result of pannus. Corneal opacity sometimes develops during the course of scrofula or chronic malaria.

Staphyloma.—Staphyloma, a bulging of the cornea, usually is a sequence of weakening of the cornea by deep ulceration, particularly in connection with gonorrheal ophthalmia.

Yellow Sclerotics.—Yellow discoloration of the sclerotics occur in jaundice from hepatic disorder. Small, circumscribed, yellowish patches, pinguecula, are innocent growths springing from the ocular conjunctiva.

Bluish Sclerotics occur in connection with chlorosis, in which they contrast markedly with the greenish discoloration of the skin, and also in nephritis and Addison's disease.

Scleritis.—In inflammation of the sclerotic coat of the eye small bluish or purplish elevations are left upon the sclerotics.

The Iris

The iris is a circular colored membrane suspended behind the cornea. This membrane is made up of circular and radial muscle fibers. The circular muscle fibers are controlled by the third nerve; the radial by branches of the cervical sympathetic. Pigment is deposited in the stroma cells. In the center is a circular opening called the pupil.

The function of the iris is to regulate the amount of light admitted to the posterior chamber by increasing or diminishing the size of the pupil. In the normal individual excess of light causes diminution in the size of the pupil. Conversely, insufficient light causes the pupil to dilate.

The pupil should be examined as to size, shape, and reaction to light and accommodation. The reaction to light is produced by shading the eye and then exposing it to light with a pocket flash light.

Consensual Light Reflex.—If during the examination for the light reflex in one pupil, the pupil of the opposite eye is observed, it will be found to react along with the pupil of the exposed eye. This phenomenon constitutes the consensual light reflex, and is due to the transmission of an impulse across the fibers which connect the two third nerve nuclei.

The reaction to accommodation is produced by directing the subject to look at a distant object and then at a near object. The pupil should dilate when accommodating for distance and contract when viewing the near subject.

Partial or total blindness will diminish or abolish these reactions.

Argyll-Robertson Pupil.—Abolition of the light reflex in one or both eyes with retention of the reaction to accommodation constitutes the Argyll-Robertson pupil, which is occasionally found in disseminated sclerosis, and very frequently in tabes dorsalis and paresis. Marina has shown this type of pupillary reaction to be caused by degenerative changes in the ciliary ganglion. In this pupil the pupillary margins are very frequently irregular, while the pupils are often somewhat myopic (spinal myosis) from disease of the cervical cord. Ultimately in tabes and paresis the pupil becomes immovably fixed, reacting neither to light nor to accommodation.

An opposite pupillary reaction, the pupil reacting to light, but failing to react to accommodation is often seen as a sequence of postdiphtheritic paralysis.

Hippus.—Rapid, rhythmic, clonic contractions of the sphincter pupillæ producing winking movements of the iris which are so gross as to be visible to the unaided eye are frequently demonstrable in disseminated sclerosis, and more rarely in hysteria, incipient acute meningitis, and epilepsy.

Pupillary Unrest.—This phenomenon, which is a normal physical finding, consisting of a regular narrowing and widening of the pupil, is so fine that it can only be demonstrated by means of the aid of a magnifying lens with the pupil brilliantly illuminated. Abolition of this normal pupillary unrest is one of the earliest signs of tabes dorsalis and paresis.

Myosis.—Contraction of the pupil may result from irritative or destructive lesions. Irritative myosis is noted in the early stages of cerebral hemorrhage, in incipient brain tumors before sufficient pressure has been exerted upon the third nerve to cause paralysis, and in early acute meningitis and encephalitis. Paralytic myosis is seen in tabes dorsalis, the late stages of tabes of the cervical cord, and syringomyelia of this portion of the cord, leading to destruction of the pupil-dilating fibers.

Mydriasis.—Irritative mydriasis is often due to irritation of the pupil-dilating center in the cervical cord from congestion, spinal meningitis, or tumor. Paralytic mydriasis may signify paralysis of the sphincter pupillæ, caused by disease of the third cranial nerve or ciliary ganglion, increased intracranial pressure from brain tumor, or glaucoma.

Irregular and fixed pupils are present in brain or spinal cord lesions, and in adhesions of the iris to cornea or lens.

The color of the pupil is usually black, but scars on the cornea, opacity of the lens, or purulent inflammation may cause the pupil to appear grayish white, yellowish white, or pinkish.

The fundus should be examined with the aid of an ophthalmoscope, noting color, condition of retinal vessels, and macula. These examinations are best conducted in a dark room with pupils dilated. Students should receive this instruction from an ophthalmologist in an eye clinic.

THE NOSE

The size and shape of the nose varies within wide limits in the normal subject. Variations in size and shape in the adult are of considerable importance.

Shape.—The shape of the nose is altered by a tumor growing within the nasal cavities or from the adjacent bones of the face. In cretinism and myxedema the nose is flattened and negroid. In syphilis in certain instances the nasal bridge is destroyed with the production of a characteristic deformity, the saddle nose.

Color.—The color of the nose varies in the normal subject with heat, cold, and emotion.

Redness of the nose, aside from being commonly associated with a history of chronic alcoholism, is observed in lupus erythematosus, in circulatory disturbances, chronic digestive disorders, and in amenorrhea.

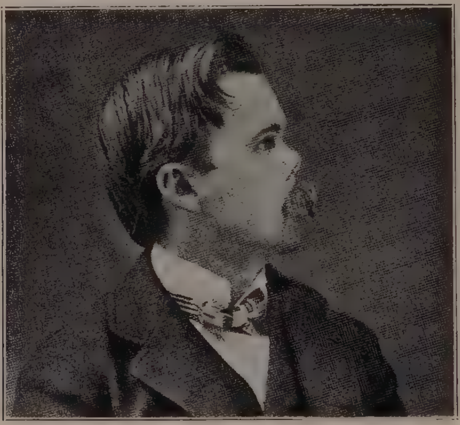


Fig. 31.—Saddle-nose. (From Eisendrath.)

Cyanosis is observed in old people, in cardiac decompensation, and acetanilid poisoning.

Nasal discharge is common in cold weather and after weeping. The character of this discharge is thin, mucoid, and clear. Bloody discharge is frequent, especially in children, but should be especially noted when associated with injury of the head. It frequently occurs in typhoid fever.

Internal examination of the nose should include color of the mucous membrane, size and shape of the turbinate, bones, and position of the septum. Red congested mucous membrane is usually seen in the common cold. Enlarged turbinates often obstruct respiration. Perforation of the septum is often the result of syphilis.

Internal examination of the nose and accessory sinuses should be taught by the specialist in nose and throat diseases.

CHAPTER IX

INSPECTION OF THE MOUTH AND THROAT

THE LIPS

The color, size, shape, and tonus of the lips, especially when these have recently changed, deserve careful consideration. Eruptions and scars should also be carefully noted.

Pallor of the lips suggests but does not prove the presence of anemia.

Cyanosis, or blueness of the lips, if not due to the ingestion of large doses of coal-tar products, is indicative of heart disease or pulmonary disease of an obstructive nature, as emphysema and pneumonia.

Parted lips, when dry and cyanotic indicate the dyspnea of cardiac or pulmonary disease. Parted lips in a child with a small, pinched nose are suggestive of adenoid vegetations in the nasopharynx.

Loose, pendulous lower lip accompanies chronic bulbar palsy, and less frequently is seen with diphtheritic palsy.

Herpes of the lips, herpes labialis, occur with pneumonia and the common cold, occasionally with malaria, typhoid fever, and other febrile affections.

Enlargement of the lips accompanies angioneurotic edema, local abscess formation, phlegmonous inflammation, and obstruction of the lymphatics draining the lips, *macrocheilia.*

Rhagades, or fissures of the lips, usually affect the lower lip near its center, in cold dry weather. Similar fissures developing upon the lips near the angle of the mouth in a child are suggestive of hereditary syphilis.

Mucous Patch.—Flat, whitish sores near the angles of the lips with sharply defined borders, are mucous patches of syphilis.

Chancre.—An indurated sore on the lip, particularly when developing in a young person, and associated with enlargement of the associated lymph glands, suggests the initial lesion of syphilis.

Epithelioma.—A chronic irregular ulcer at the mucocutaneous junction of the lower lip in a person past middle life, with en-

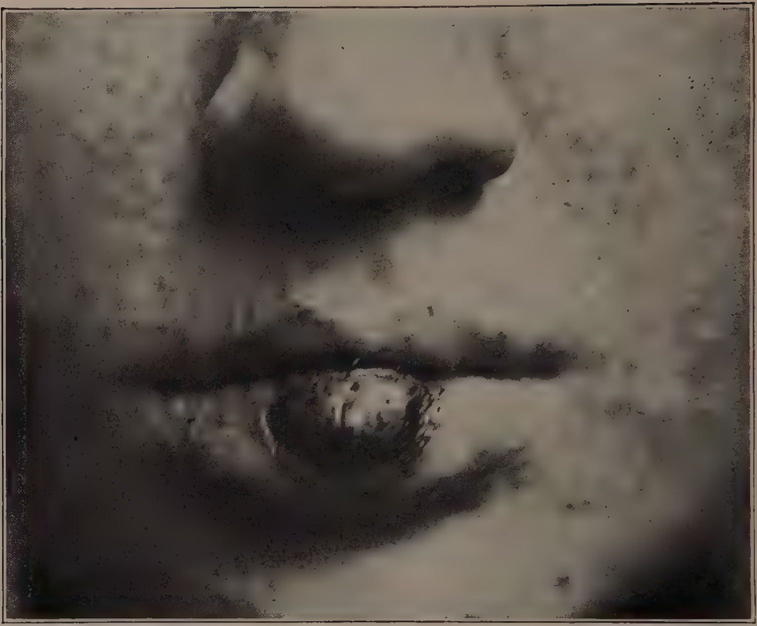


Fig. 32.—Chancre of lip, with associated macular eruption. (Varney, from Sutton, *Diseases of the Skin.*)

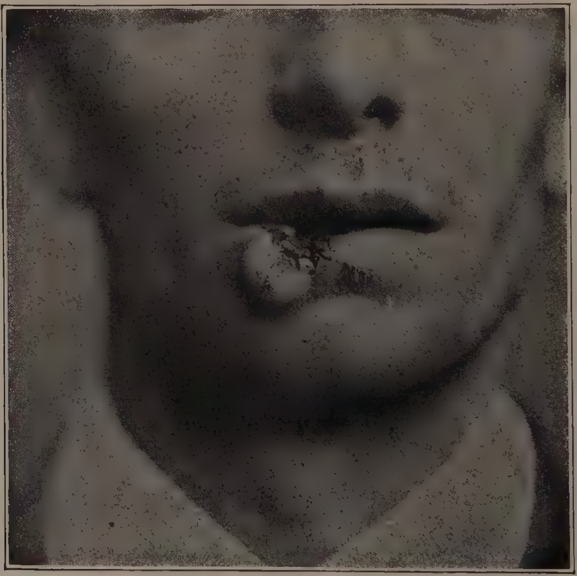


Fig. 33.—Prickle-celled carcinoma of the lower lip in a young man, which arose after treating a clinically benign lesion with caustic pastes. (Gilchrist's collection.) (From Hazen.)

largement of the lymph glands at the angle of the jaw, is suggestive of epithelioma.

Harelip is recognized as a vertical slit or cleft in the upper lip on one or both sides of the median line. The cleft may be small and confined to the lip, or may be associated with cleft palate, club-foot or other deformity.

Noma, or cancrum oris, is recognized as a gangrenous mass of tissue involving the lip and adjacent surface of the cheek accompanied by a very foul odor. Occurring after measles and diphtheria, it is frequently a sequence of ulcerative stomatitis.

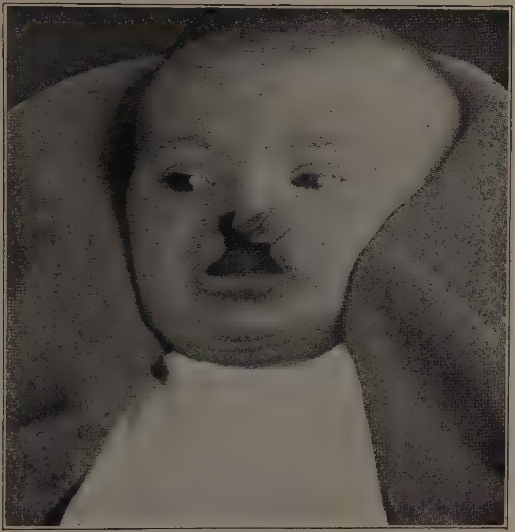


Fig. 34.—Double harelip and cleft palate. (From Eisendrath.)

THE BREATH

The odor of the breath is frequently altered by food or drink, but unusual odors, or odor of food or drink remaining an unusual time on the breath, should be noted.

Foul Breath.—A foul breath may be caused by local disease of the buccal cavity, notably carious teeth, pyorrhea alveolaris, ulcerative or gangrenous stomatitis, follicular tonsillitis, and by nasopharyngeal ulceration. Gangrene of the lung is attended by marked fetor of the breath. A moderately offensive breath is frequently noted in the presence of chronic gastritis, intestinal toxemia, and chronic constipation.

Uremic Breath.—In uremia the breath has a urinous or ammoniacal odor.

Diabetes.—Diabetes mellitus imparts a sweetish, fruity odor to the breath, the acetone breath.

THE TEETH

Primary dentition usually follows a definite order. The lower central incisors appear about the seventh month, the four upper incisors at the tenth month, the premolars at the thirteenth month, the canines at the twenty-first month, and the molars at the twenty-seventh month. From the sixth to the seventh year the first permanent molars appear, followed by the upper and lower incisors, about the eleventh year the second bicuspids and canines, about the twelfth year the second molars, and the last molars from the eighteenth to the twenty-fifth year.

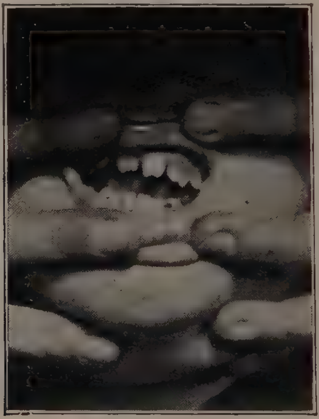


Fig. 35.—Hutchinson's teeth. There was also present an interstitial keratitis. (C. Morton Smith, from Sutton, *Diseases of the Skin.*)

Premature and **delayed** dentition posses diagnostic significance. The former suggests hereditary syphilis, while the latter accompanies rickets, cretinism, and disorders of nutrition.

The shape and size of the teeth and the state of repair and dental hygiene may be of significance.

Early Decay.—In children early decay of the teeth occurs in association with rickets and gastrointestinal disorders. In adults carious teeth occur in pregnancy and diabetes mellitus as well as in chronic phosphorus poisoning.

Darkened teeth are usually devitalized and when a suspicion of focal infection occurs, they should be x-rayed for evidence of apical absorption or abscess.

Loosening of the teeth, with spongy bleeding gums, occurs in scurvy, mercurial poisoning, and in pyorrhea alveolaris.

Hutchinson Teeth.—In hereditary syphilis the upper central incisors may present each a notch in its free border. These teeth are small and separated by distinct intervals. The permanent and not the deciduous teeth are affected.

Grinding of the teeth during sleep in children is observed in connection with rickets and derangements of digestion.

THE GUMS

The gums should adhere closely to the teeth, should be firm, and of a pinkish color, varying somewhat in individuals. The gums usually reflect the state of dental hygiene.

Blue Line.—A blue line on the margin of the gum is indicative of chronic lead poisoning. In the early stages of the intoxication the line is not continuous, but occurs as a series of blue dots at the base of the teeth. The line may extend along the entire length of the gum, or may be limited to the bases of a few of the front teeth in either jaw.

In chronic copper poisoning a blue or greenish line develops along the roots of the teeth.

Red Line.—A red line along the gingivodental margin occurs with pyorrhea alveolaris, gingivitis, frequently in diabetes, and it is said, in tuberculosis.

Spongy Gums.—In ulcerative stomatitis the gums are swollen, spongy, of deep red or purple color, with a line of ulceration adjacent to the incisors, sometimes extending to all the teeth.

In scorbutus the gums are spongy, bleed easily, and the teeth are loosened. In mercurial poisoning the gums are spongy, there is excessive salivation, and fetid breath.

In pellagra the gums are spongy, and assume a cerise color.

Epulis.—An epulis is a small, soft tumor springing from the gums or alveolar process of the superior maxillary bone. It is usually a giant-cell sarcoma.

THE TONGUE

The tongue is a double muscle united in the median line. The size, method of protruding, color, moisture, papillae, and scars should be noted. Both the upper and under surfaces should be examined.

Size of the Tongue.—*Hypertrophy* of the tongue occurs in infants as a congenital condition, the tongue reaching such enormous size that it cannot be contained in the mouth. *Acquired hypertrophy* of the tongue is seen in acromegaly, myxedema, and cretinism, in acute glossitis, and as a result of lymphatic obstruction, macroglossia.

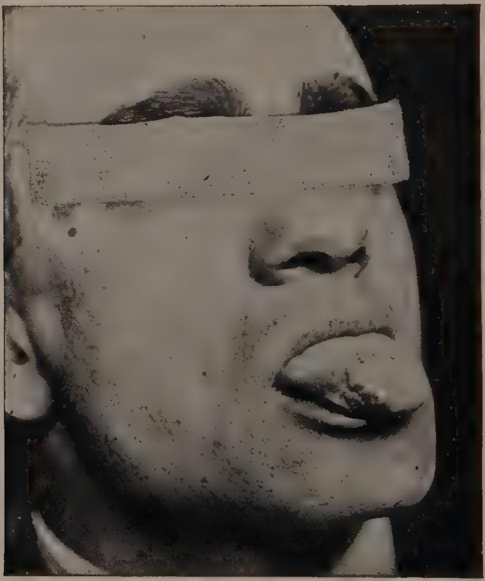


Fig. 36.—Tuberculous ulcer of tongue (tubercle bacilli demonstrated in section). (Morrow, Miller and Taussig, from Sutton, *Diseases of the Skin.*)

Atrophy of the tongue occurs as a part of glossolabiolaryngeal paralysis. Unilateral atrophy of the tongue may accompany facial hemiatrophy, and may occur as a result of hemorrhage or tumor developing in close proximity to the hypoglossal nucleus.

Movements of the Tongue.—The manner in which the tongue is protruded upon request as well as the integrity of its movements during mastication and speech should be carefully noted. Thus in nervous and neurasthenic subjects the tongue is protruded quickly upon request, whereas in typhoid states the protrusion is very slow and tardy.

In unilateral paralysis of the tongue, accompanying hemiplegia or unilateral hypoglossal palsy the tongue deviates from the median line when protruded. In bilateral paralysis of the tongue, as a result of bulbar paralysis, or symmetrical lesions of the cortex or supranuclear tracts, the tongue lies upon the floor of the mouth and cannot be protruded.

Inability to perform the finer movements of the tongue concerned in mastication and speech is an early sign of glossolabiolaryngeal paralysis, or true bulbar palsy. A similar impairment of the movements of the tongue accompanies pseudobulbar paralysis, a state in which the central lesion is not situated in the medulla, but in the lingual fibers from the cortex above the medulla. This condition of pseudobulbar palsy is not accompanied by atrophy of the tongue, which only occurs in true bulbar palsy, or glossolabiolaryngeal paralysis. The absence of lingual atrophy in the former indicates that the causative lesion is in the upper neurone, above the nucleus.

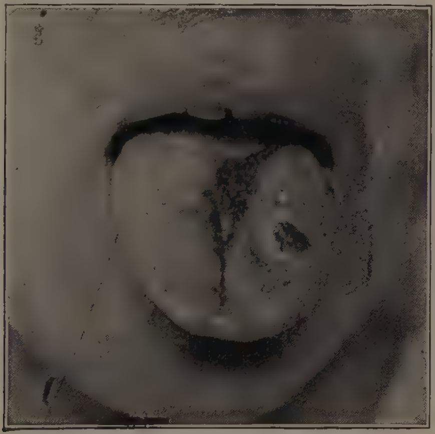


Fig. 37.—Gumma of tongue. (From Sutton, *Diseases of the Skin.*)

Tremor.—A coarse or fine tremor of the tongue upon protrusion accompanies many organic nervous diseases and typhoid states, and exhausting fevers. In organic nervous involvement the tremor is constant, whereas in typhoid states it only develops upon protrusion of the tongue.

Spasm.—Tonic spasm of the tongue accompanies Thompsen's disease, or myotonia congenita. A similar tonic spasm of the tongue occurs from reflex irritation of the trigeminal nerve. Clonic spasm of the tongue is noted in connection with chorea, epilepsy, puerperal melancholia, multiple sclerosis and paresis.

Ulceration.—Ulceration of the dorsum of the tongue may be indicative of simple ulceration, tuberculosis, syphilis, or carcinoma.

Simple ulceration results from local trauma or irritation. Not infrequently in young children an ulcer of the frenum is noted, resulting from the irritation of the sharp edges of the lower central incisor teeth.

A *tuberculous ulcer* of the tongue may be oval, linear, or stellate. The surface of the ulcer is pale and uneven, covered with grayish exudate, presenting no evidences of acute inflammation. It is usually accompanied by tuberculosis of the cervical lymphatic glands.

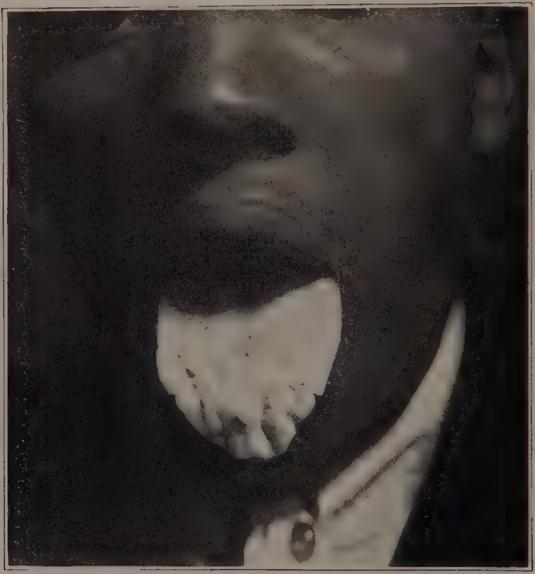


Fig. 38.—"Cobblestone tongue" due to gummous deposits two years after infection. (From Hazen.)

A *syphilitic ulcer* of the tongue produces a chronic dissecting glossitis, characterized by multiple fissures situated principally upon the lingual edges, but crossing the dorsum of the tongue in various directions. The cervical lymph glands are commonly enlarged.

A *carcinomatous ulcer* of the tongue is solitary, with induration of the surrounding tissues, somewhat simulating a chancre; but the ulcer does not disappear under antisyphilitic therapy. The carcinomatous ulcer is apt to develop in elderly persons, and the accompanying glandular enlargement appears more tardily than in the other lingual ulcers.

Geographic Tongue.—In the geographic tongue there are one or more patches upon the dorsum in which the surface epithelium has desquamated, the patches extending at the periphery while healing at the center, pursuing a circinate course over the dorsum, two or more patches frequently coalescing. Occurring in undernourished children and adults, the geographic tongue possesses little significance.

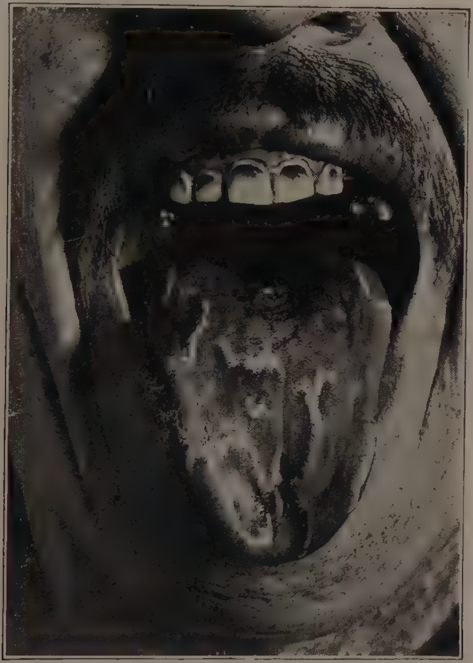


Fig. 39.—Geographic tongue, showing typical case. A very unusual photograph. (MacKee, from Sutton, *Diseases of the Skin.*)

Leucoplakia.—In leucoplakia irregularly shaped plaques of thickened epithelium appear upon the dorsum of the tongue. The plaques are smooth, pale, slightly elevated above the surrounding surface of the tongue, one to two centimeters in diameter, and nonulcerative. Their recognition is important in that they have become the starting point of carcinoma of the tongue.

Smoker's Patch.—In persons who use tobacco to excess a round or oval patch is sometimes encountered upon the dorsum of the tongue near the tip, slightly elevated, red or pearly in color, smooth, and with no tendency to ulceration.

Cysts.—Mucous and blood cysts occasionally develop in the tongue. Rarely the cysticercus cellulosæ, the larva of the tænia solium may produce a cyst upon the under surface of the tongue, or an echinococcus cyst may be encountered in this region. Also a ranula, due to obstruction and dilatation of the ducts of Blandin-Nuhn's glands may be found on the under surface of the tongue near the tip.

Thrush.—In thrush the dorsum of the tongue is covered or studded with small, white flakes, resembling closely deposited flakes of coagulated milk, but differing from them in that they cannot be wiped off; and, if removed, leave bleeding points.

Indentation of the edges of the tongue by the teeth, is noted during prolonged fevers when the hygiene of the oral cavity is not properly practiced. In pellagra there are similar indentations in the deep red border of the tongue occurring with this disease.

Pellagra.—In pellagra the tongue presents a fiery red border and tip, showing indentations corresponding to the teeth with which it is in contact. Frequently small circumscribed sloughs are encountered upon the borders of the tongue, corresponding to areas of epithelial denudation. The gums are spongy and of a cerise color, while the mouth is the site of a stomatitis of variable intensity. The condition of the tongue and buccal mucosa is intensely painful.

Dryness of the Tongue.—The tongue in health is kept moistened with the salivary secretions and the buccal secretions. When this secretion is inhibited during acute fevers, the administration of atropine, or from excessive loss of body fluids incident to prolonged diarrhea and profuse hemorrhage the tongue is abnormally dry, and not infrequently covered with a thick brown coat.

Color.—The ingestion of various chemicals and drugs and certain diseases alter the color of the tongue. The tongue is white following the ingestion of mercuric chloride, ammonia, sulphuric acid and phenol. The ingestion of caustic potash or soda causes reddening of the tongue with evidences of destructive action of these substances. Hydrochloric and nitric acids color the tongue yellow.

In Addison's disease and purpura hemorrhagica the tongue not infrequently exhibits small nonelevated purple spots; while xanthelasma produces yellowish, slightly elevated dots along the margins of the tongue.

In scarlatina the tongue is bright red, the filiform papillæ contrasting sharply with the slight white furring of the tongue, the *strawberry tongue.*

Scars on the tongue are often the result of biting during epileptic seizures, and are of considerable importance, especially when suspicious cases deny having suffered from the disease.

Sublingual ulcerations are often noted in whooping cough. The yellow pigmentation of jaundice is quite evident on the under surface of the tongue.

The ducts of the sublingual and submaxillary glands, while very difficult to see in the normal individual, often reveal their location by spurting a fine stream of saliva.

THE BUCCAL CAVITY

The buccal cavity should be examined with the aid of a wooden tongue depressor, care being taken to examine the entire surface as to color, moisture, eruptions, scars, and the opening of the parotid duct at the level of the first upper molar tooth.

Color.—The buccal mucosa is pallid in anemic states, is excessively red during local inflammation as in catarrhal stomatitis, and is bluish in cyanosis and argyria.

Moisture.—Excessive moisture of the buccal cavity, incident to overactivity of the salivary and buccal glands accompanies local inflammation, following the ingestion of massive doses of the iodides and mercurial salts, during the early stages of smallpox and typhus fever, occasionally during pregnancy, and during convalescence from typhoid fever.

Dryness of the buccal cavity, or xerostomia, owing to temporary arrest of the salivary and buccal secretions occurs during acute febrile diseases, diabetes mellitus, in mouth breathers and lesions of the pons and medulla affecting the integrity of the nervous mechanism of the salivary glands.

Eruptions.—In variola and varicella vesicles may appear upon the buccal mucosa, similar vesicular eruptions accompanying herpes buccalis and aphthous stomatitis. Measles is accompanied by pathognomonic lesions upon the mucosa, *Koplik's spots.* These are minute red spots with a bluish-white center, occurring upon the inner surface of the cheek opposite the molar teeth. The number of the spots varies; there may be only one or two or the mucosa may be fairly studded with them. The spots occur early, disappearing with the inception of the exanthem.

Mucous Patch.—The mucous patch of syphilis is frequently encountered upon the buccal mucosa. In all suspicious cases a careful search of the mucous lining of the cheek should be made for these lesions.

Noma, cancrum oris, or gangrenous stomatitis, develops as an indurated spot upon the mucous lining of the cheek near the angle of the mouth, later involving the entire thickness of the buccal wall, the gangrenous tissue emitting an especially foul odor.

Scars on the buccal surface, like scars on the tongue, may be evidence of biting the cheek during an epileptic seizure. Healing of accidental or surgical trauma and the ulcerations noted above may also produce scars.

The parotid duct appears as a small opening, often surrounded by a red areola. Stoppage of this duct may be evidenced by excessive dryness of the buccal membrane on the affected side. Tumors and calculi at the opening of the duct may be overlooked if the cheek is not pressed away from the teeth with a tongue blade.

THE PHARYNX, UVULA, HARD AND SOFT PALATE

In examining the pharynx the tongue should be depressed with a wooden spatula or other type of tongue depressor, while the patient is instructed to utter the word "AH," which lowers the base of the tongue, permitting a good view of the posterior pharyngeal wall.

Pharyngeal or gag reflex is the production of retching by irritating the posterior pharyngeal wall with a finger or tongue depressor. This reflex is often absent, especially in asthenic individuals.

Color, scars, eruptions, movements of the soft palate and uvula, and color, scars, and eruptions of the hard palate should be noted at this time.

Redness.—Abnormal redness of the pharyngeal wall accompanies acute inflammation of the pharynx, which may be primary, or occur with the acute exanthematous fevers, or acute infectious disease, as influenza and erysipelas.

Eruptions.—The eruptions of variola, varicella, and of herpes buccalis are often distributed generally over the pharyngeal wall.

Ulceration of the pharyngeal wall is indicative of tuberculosis, syphilis, or typhoid fever.

Bulging of the posterior pharyngeal wall, either in the median line or laterally, occurs in postpharyngeal abscess, which is often due to tuberculous disease of the cervical vertebræ.

Elongated Uvula.—Elongation of the uvula may occur from inflammation of the adjacent pharyngeal mucous membrane, but it may also be a part of the general edema incident to cardiac or renal disease.

Perforation of the soft palate is usually of syphilitic origin.

Paralysis of the soft palate may be unilateral or bilateral. Paralysis of the palate is detected by observation of its movements while the patient speaks, at which time the normal palate moves upward. If this normal mobility of the palate is lost on one side, the paralysis is unilateral; if both sides remain immobile, the paralysis is bilateral. Bilateral palatal paralysis is not infrequently attended by regurgitation of fluids through the nose upon the attempt to swallow. Paralysis of the palate may be part and parcel of glossolabiolaryngeal paralysis, may depend upon cervical caries, or may be due to diphtheritic paralysis.

Cleft palate is usually associated with harelip. It may be partial or complete. Eruptions on the soft and hard palate occur in scarlet fever, measles and varicella.

THE TONSILS AND FAUCES

The tonsils lie between the anterior and posterior fauces. They are often submerged and pressure on the neck opposite this location will serve to bring them into the field of vision, as will exciting the gag reflex by placing the tongue depressor rather far back on the tongue.

The fauces should be examined for moisture, eruptions and exudate.

Eruptions on the fauces are usually due to scarlet fever or measles; exudative membrane or pseudomembrane to streptococcus infection or diphtheria.

The tonsils are usually well developed in infants and children, becoming smaller in adult life, and almost disappear in most aged people.

One should note their size, shape, color and crypts.

Inflammation.—A moderate grade of tonsillar inflammation resulting in painful deglutition, accompanies most of the acute exanthematous fevers. In acute follicular tonsillitis the tonsils are moderately enlarged, red, and studded with minute yellowish dots, corresponding to plugs of mucus, epithelium and bacteria which can be squeezed from the tonsillar crypts.

Chronic simple enlargement of the tonsils, in which the two bodies may almost meet in the median line, occurs occasionally in childhood.

Pseudomembrane.—A pseudomembrane upon the tonsil, perhaps involving the fauces and pharyngeal wall as well, if not diphtheria, is apt to be due to streptococcic inflammation or scarlatina.

Ulceration.—Ulceration of the tonsil is due to tuberculosis, syphilis, or, if in an elderly person, to carcinoma, or in a younger subject to sarcoma.

Peritonsillar Ulceration (Vincent's Angina).—In this disease which is a unilateral affection, there is ulceration of the peritonsillar tissues, with a variable amount of yellowish exudate covering the tonsil. There is marked swelling of the submaxillary lymph glands.

Peritonsillar abscess causes considerable swelling behind the tonsil, usually forcing the tonsil forward, sometimes almost occluding the passage and making deglutition and even breathing difficult. It is often associated with acute tonsillitis, and is usually unilateral.

THE EPIGLOTTIS AND VOCAL CORDS

The examination of the epiglottis and vocal cords should be taught by the laryngologist in a throat clinic where mirrors and other apparatus are at hand.

Color, edema, and ulceration of the epiglottis should be noted.

Color.—In the anemias the epiglottis is pale, while in inflammations it may be bright scarlet.

Edema occurs in diphtheria, scarlet fever, and is associated with allergic states.

The vocal cords should be examined for movements during phonation, scars and eruptions.

Paralysis of the vocal cords may result from diphtheria, tuberculosis, syphilis, or disease or injury to the recurrent laryngeal nerves.

Ulcerations of the vocal cords may be due to tuberculosis or syphilis.

CHAPTER X

INSPECTION OF THE NECK

Shape.—The shape of the neck varies with the habitus of the subject. The long thin neck is characteristic of the asthenic type, while the short thick neck characterizes the sthenic or hypersthenic type.

Certain variations in the shape of the neck characterize certain diseases. Thus, a short, thick neck suggests hypertrophic emphysema, and is a constant accompaniment of the barrel chest of this disease. A long, slender neck, on the other hand, is frequently observed in phthisical patients.

Skin.—The skin should be observed for scars, eruptions, color, and evidence of weathering.

Movements.—Movements of the neck in turning the head from side to side and backward and forward should be carefully observed, and any restriction noted.

Rigidity.—This may be caused by tuberculous disease of the cervical vertebræ or spasm of the muscles of the neck. Tender, enlarged cervical glands, boils or carbuncles may cause the patient to hold the neck rigid. As previously stated, retraction and rigidity of the neck occur in meningitis, tetanus, and strychnine poisoning. Rigidity of the neck, with fixation of the head, is also observed as a result of arthritis deformans, spasmodic torticollis, and to scars from extensive burns of the neck.

Musculature.—General muscular development and development of certain groups of muscles may be the result of exercise, occupation or disease.

Prominent Sternomastoids.—Abnormal prominence of both sternomastoid muscles is usually a sign of long continued dyspnea, due to pulmonary or cardiac disease. An undue prominence of one sternomastoid may be caused by spasmodic torticollis, a tumor, cyst, or abscess of the muscle.

Muscular atrophy may be the result of wasting illness, accident, or surgical operation involving injury to muscle or nerve supply.

Spasm of the neck muscles, especially the sternomastoid and trapezius, frequently occurs either as the result of disease or pressure on the spinal accessory nerve by tumors, enlarged glands, or disease of the cervical spine. Congenital spasm is caused by congenital shortening of the sternomastoid muscle.

THE LARYNX AND TRACHEA

The larynx and trachea should be observed for position, prominence, and movements during breathing and swallowing.

Deflection of Larynx and Trachea.—The larynx and trachea, the latter overlaid by the thyroid gland, occupy the median line of the neck. Deflection of these structures to one or the other side may be due to atrophy of the muscles on one side of the neck, to tumor or aneurysm in the adjacent tissues, or to disease of the thoracic viscera. Of the last named factors, fibroid phthisis is very important, the structures being deflected toward the side of the cirrhotic lung.

Movements of the Larynx and Trachea.—Marked inspiratory descent of the larynx occurs in laryngeal stenosis. Normally the larynx descends slightly during inspiration and rises to a similar degree during expiration. When this mobility is abolished in a dyspneic patient the cause of the dyspnea is below the larynx as, for instance, pressure on a bronchus by enlarged glands or aneurysm.

THE GLANDS

Thyroid Gland.—The thyroid gland, located below and to each side of the thyroid cartilage, is not visible in the normal subject, but enlargement is very common, due to simple hypertrophy, cysts, over activity, as in exophthalmic goiter, or from malignant or benign tumors. This enlargement may be of either lobe or isthmus.

Diminution in the size of the gland may be evidenced by a depression at the normal site of the gland.

Prominence of cricoid cartilage is often striking but of little significance.

Lymph Glands.—The lymph glands of the neck are not visible in the normal subject, but their location should be borne in mind. A chain of glands lies both anterior and posterior to the sternomastoid muscle at the angle of the jaw, and in the submaxillary and occipital regions.

Enlarged Glands.—Enlarged lymph glands in the cervical region may have a varied significance, the significance varying with the location of the glands involved and with the state in which they are found; namely, whether hard or soft and fluctuating, and whether single and individual or matted together in a mass. Among the causes of glandular enlargement in this region may be mentioned the following conditions.

The lymph glands at the angle of the jaw may enlarge from follicular tonsillitis, diphtheria, scarlatina, measles, German measles, varicella and smallpox; also in erysipelas, glanders, whooping cough, and retropharyngeal abscess. In these conditions the glands are acutely tender for a period and usually undergo resolution without abscess formation.

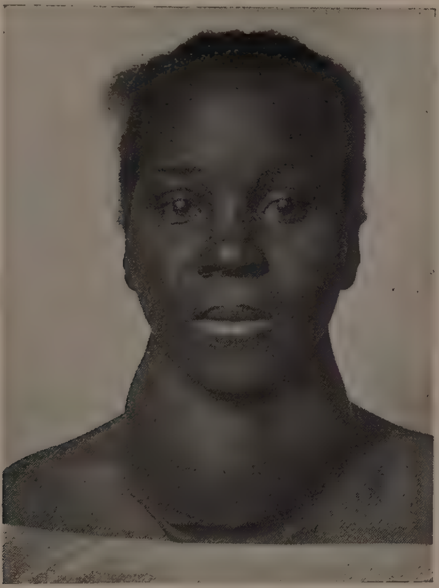


Fig. 40.—Goiter. (From Woolley.)

The submental glands, just below the chin may enlarge as a result of carious teeth, stomatitis, syphilis, mumps, and cancer of the lower lip or anterior portion of the tongue. This group of glands is often enlarged in cases of actinomycosis.

The parotid lymph glands enlarge in mumps, and diseases of the upper pharynx and skin of the face, as well as in malignant disease of the parotid gland.

The occipital glands are enlarged in German measles, often the only group attacked in this disease. Enlargement of this group of glands is also a valuable sign of syphilis, and occurs also in cases of pediculosis of the scalp.

Tuberculosis of the cervical glands causes glandular enlargement, particularly posterior to the sterno-mastoid muscle and in the glands under the jaw. The glands tend to become adherent to the cutaneous structures and often suppurate.

Hodgkin's disease causes glandular enlargement in the lymph glands of the neck.

Lymphatic leucemia is a cause of enlargement of the cervical lymph glands.

Enlargement of the lymph glands above the left clavicle occurs in cancer of the stomach.

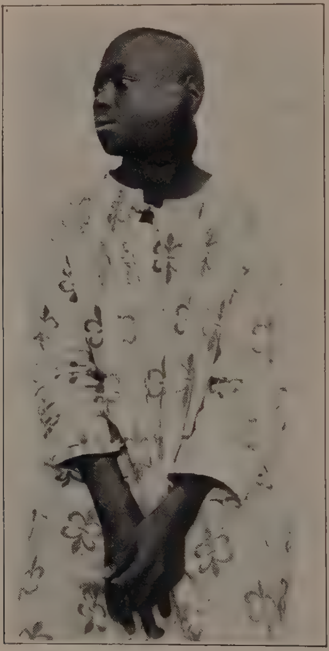


Fig. 41.—Hodgkin's disease.

The condition of the glands and the duration of the enlargement possess diagnostic significance. Thus, acute painful cases of short duration are probably due to a tonsillitis, or the exanthemata. Chronic cases, of long standing, may be due to tuberculosis, syphilis, or Hodgkin's disease. In tuberculosis the glands are matted together with tendency to suppurate. In syphilis they are hard and small. In Hodgkin's disease the glands are enlarged, but remain separate, and do not tend to suppurate.

Salivary Glands.—The location of the salivary glands at the angle of the jaw and in the submaxillary space should be observed.

Tumors or indurations due to blocking of the salivary ducts, malignancy, or acute epidemic infections, such as mumps, may affect any or all of these glands.

The pulsation of the carotid artery can usually be seen just anterior to the lower third of the sternomastoid muscle. These pulsations of the right and left carotid should be compared as to force, rate and rhythm. The size of the artery should also be noted.

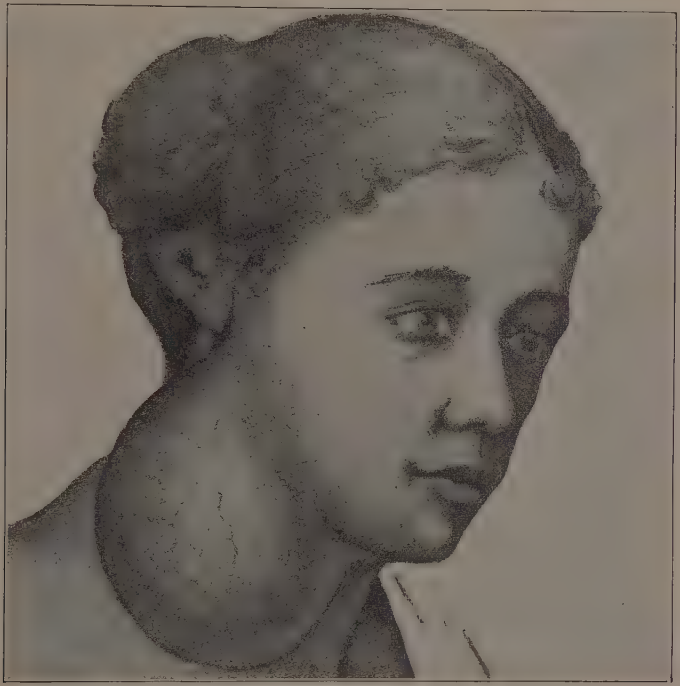


Fig. 42.—Branchial cyst. (From McFarland.)

Variations of force occur in aneurysm of the aortic arch and innominate artery. In aneurysm of the transverse portion of the aortic arch the right radial pulse is stronger than the left, while in aneurysm of the innominate artery the left is stronger than the right. Violent throbbing pulsations occur in aortic regurgitation.

The veins of the neck vary considerably in size and location. At times the anterior jugular vein may cross the carotid artery and the pulsations of this artery may be transmitted to the vein, giving the appearance of a venous pulsation. In tricuspid regurgitation true venous pulsation occurs. Pressure on the descending vena cava from mediastinal tumors may cause marked engorgement of the veins of the neck.

Tumor masses may be seen on one or both sides of the seventh cervical vertebrae, due to a cervical rib. Cervical ribs occur in 1 to 2 per cent of the population.

Abscess.—An abscess in the cervical region is almost certainly of tuberculous origin, being the result of tuberculosis of the cervical lymphatic glands or of Pott's disease

Scars.—Scars in the neck are usually the result of cervical tuberculosis, surgical procedures, or trauma.

Branchial Cysts and Fistulæ.—Branchial cysts and fistulæ, resulting from imperfect closure of the embryonic branchial clefts, are encountered in rare instances. A branchial cyst is formed by closure of the pharyngeal and cutaneous surfaces of the cleft without closure of the intervening mesoblastic tissues. Branchial fistulæ may be complete or incomplete, depending upon the degree of fusion of the embryonic clefts, the incomplete being represented by diverticula, either external or internal, opening into the pharynx.

Ludwig's angina, a painful indurated swelling beneath the angle of the jaw, due to septic infection of the tissues surrounding the submaxillary gland, is an occasional cause of dyspnea and dysphagia which may become alarming.

Woody or ligneous phlegmon, an insidious induration of the subcutaneous tissues of the neck, involving the lateral or anterior aspect of the cervical region, is occasionally encountered. In some instances the structures are indurated from the jaw to the clavicle. The condition is attended by little pain and no fever.

CHAPTER XI

INSPECTION OF THE UPPER EXTREMITIES

THE HANDS

Careful inspection of the hands requires but a moment or two and may give a great deal of valuable information. A comparison of the right and left hands should always be made.

The color depends upon exposure to sun and thickness of the skin, as well as to the blood pigment. Chemical irritants or disease may cause roughing, irritation, or actual inflammation, examples being eczema, poison ivy, creosote poisoning, etc. Exfoliation of the skin of the palms may follow scarlet fever.

Pellagra.—The cutaneous manifestations of pellagra involve the extensor aspect of the hand and forearm, producing an eruption of an erythematosquamous type. In incipient cases, in the stage of initial erythema, the eruption resembles closely ordinary sunburn or solar erythema. In the latter evolution of the disease the superficial epithelium takes on a brown pigmentation, and desquamates in scales. Sensation is lost in the areas of desquamation. The skin covering the elbows should always be inspected for the eruption. The eruption may involve the dorsum of the foot or the face, and rarely the neck or chest.

The evidence of occupation, such as calluses, abrasions, and scars should be observed.

The shape of the hand varies with the body type or habitus. The asthenic type has a narrow slender hand with long fingers, while the sthenic or hypersthenic type has a broader hand with shorter and thicker fingers. Disease may cause considerable variation in the shape of the hand.

Spade Hand.—In acromegaly and myxedema the hand is large, with thick fingers and broad nails. In myxedema the hand is boggy, but does not pit on pressure; whereas in acromegaly the hand is hard, as the basis of the hypertrophy is osseous.

Claw-Hand (Main-en-griffe).—In amyotrophic lateral sclerosis and progressive muscular atrophy the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are flexed toward the palm. The underlying cause of this deformity is a paralysis of the lumbrical and interosseous muscles, causing the proximal phalanges to assume a state of dorsal extension, while the distal phalanges are flexed.

Hemiplegic Hand.—In hemiplegia the contractures of the arm and hand are replaced in course of time by permanent deformity. The fingers in this deformity are flexed upon the palm, the wrist is flexed upon the forearm, while the elbow is retained in a state of permanent flexion and applied closely to the side of the body.

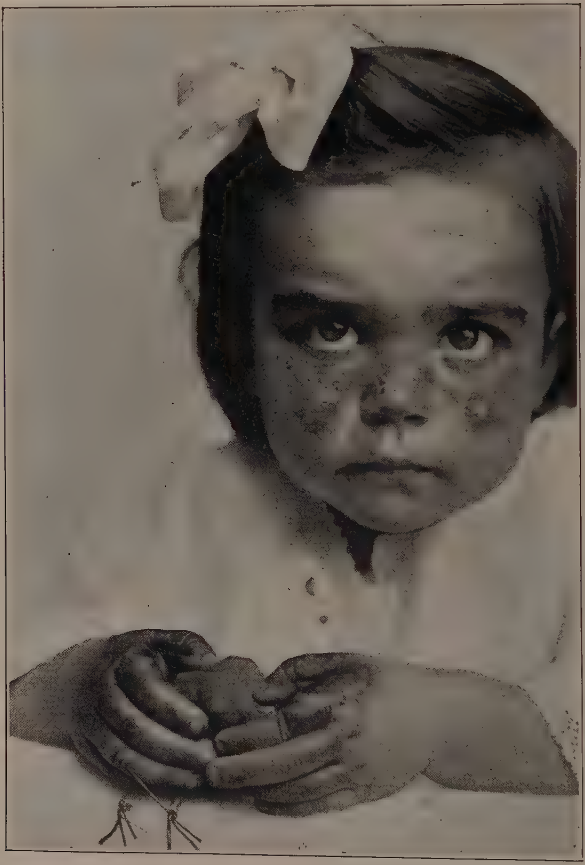


Fig. 43.—Pellagra in child less than 3 years old.

Seal-fin Hand.—In chronic gout and rheumatoid arthritis the entire hand is deflected toward the ulna as a result of spasm of the extensor muscles, imparting to the hand a fancied resemblance to the fin of a seal.

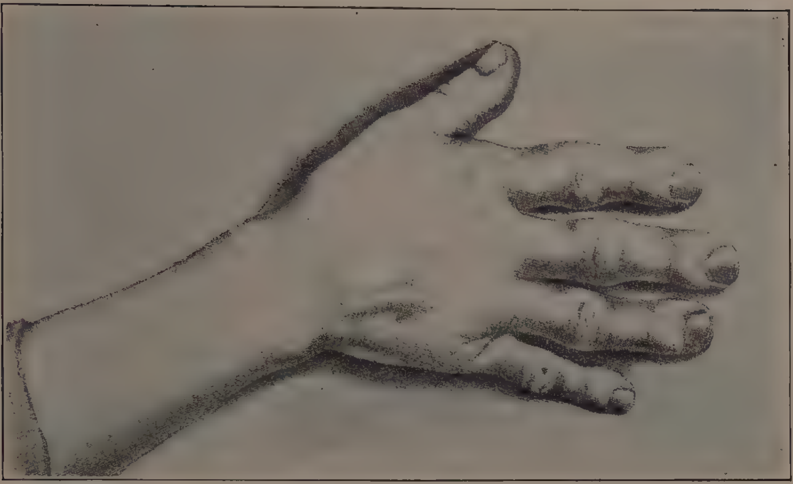


Fig. 44.—Spade hand. (From Butler.)

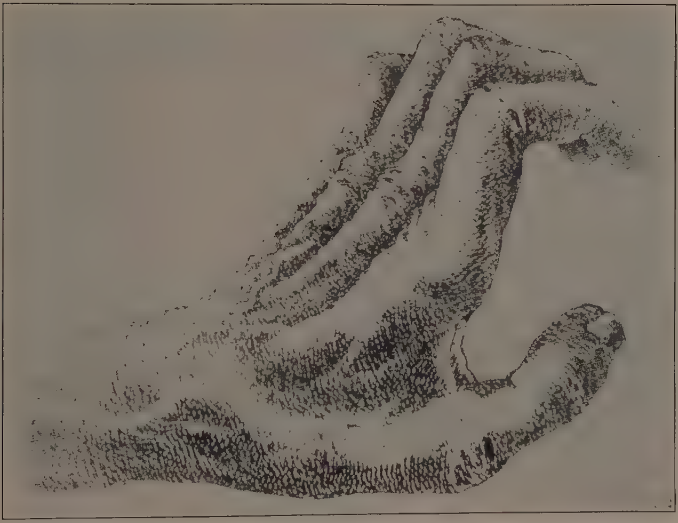


Fig. 45.—Claw hand. (Main-en-griffe.) (From Eisendrath.)

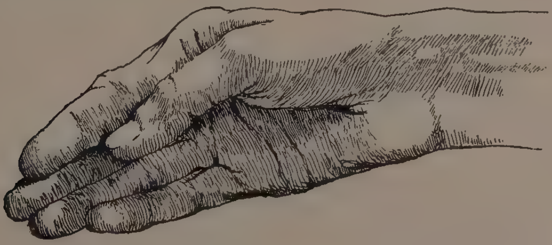


Fig. 46.—Accoucheur's hand. (From Butler.)

Ape Hand.—This type of manual deformity is the result of wasting of the thenar and hypothenar muscles in progressive muscular atrophy, causing the hand to assume a position in which the fingers and thumb are on one parallel plane.

Accoucheur's Hand.—In this type of manual deformity, which occurs in tetany, the thumb is flexed into the palm of the hand, while the fingers, flexed at the metacarpophalangeal joints and first interphalangeal joints, are extended at the second interphalangeal articulations and pressed closely upon the thumb.

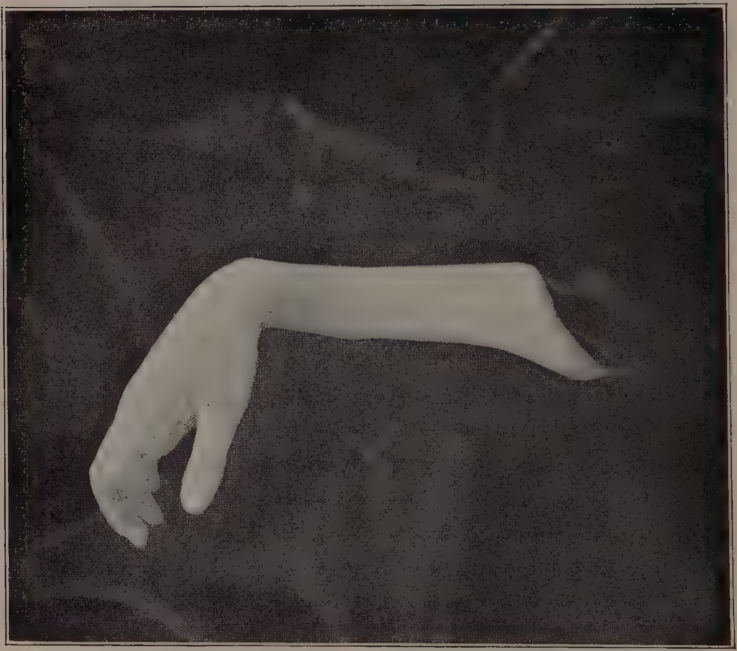


Fig. 47.—Wrist-drop. (From Eisendrath.)

Dupuytren's Contracture.—This is a permanent painless flexure of one finger of one or both hands into the palm. Most commonly the little finger alone is involved, but sometimes the ring finger or other fingers are flexed also. Dupuytren's contracture results from occupational irritations, burns, or other injuries to the palmar fascia.

Ganglion.—A ganglion is recognized as a localized swelling upon the dorsum of the hand. It is presumably caused by cystic degeneration of a synovial fringe within a tendon sheath. Ganglia are not infrequently tuberculous in origin.

Wrist-drop.—In wrist-drop the hand hangs powerless from the wrist. It is significant of neuritis or paralysis of the musculo-spiral nerve.

Movements of the hand both voluntary and involuntary should be noted. The subject should be told to extend the hands first with the fingers approximated and then with the fingers separated. Involuntary movements are generally noted during this procedure, such as tremor and spasms, as in deformities and wasting paralysis of the muscles. Opening and closing both hands and movements of the wrist joints should also aid in detecting changes of muscles or joints.

Intention Tremor.—Intention tremor is a tremor of the hand which is converted into coarse shaking movements when the patient endeavors to perform any act, such as bringing a glass of water to the lips, or holding a pen to write. Intention tremor is a cardinal sign of multiple sclerosis and is sometimes noted in hysteria.

Paralysis Agitans.—In paralysis agitans the patient is subject to a constant tremor of the hands, in which the thumb and index finger are held in close proximity to one another or in actual contact, describing a rolling movement as if they were rolling a pill (pill-rolling tremor). The tremor, in marked contrast to that of multiple sclerosis, disappears completely during voluntary movements of the hands.

Professional Spasm.—Writers, violin players, and others who constantly employ one set of muscles, are often troubled with painful spasms in the muscles used, incapacitating them for their usual occupations.

Athetosis.—This term refers to certain slow and purposeless movements of the fingers which are encountered in patients suffering with organic disease of the central nervous system.

The Nails

Detailed examination of the hands should begin with the nails. Their color, shape, thickness, whether smooth or rough, as well as the care of the nails, should be noted. Evidence of biting the nails is common in both children and adults. It usually signifies a rather irritable or mal-adjusted mental state. The skin at the root of the nails often breaks causing hang nails which become easily infected in individuals of subnormal resistance.

Pallor.—Pallor of the nails is a sign of anemia, and it is well to bear in mind the rule laid down by Stephen McKenzie, that when pressure upon the tip of the finger completely drives the blood from beneath the nail, the red corpuscles are present in only half their normal number.

Cyanosis.—Cyanosis or blueness of the nails is a sign of deficient aeration of the blood, either due to a failing heart, an obstructive pulmonary lesion, or the ingestion of coal tar products. Cyanosis appears very early under the nails and about the lips.

White spots in the nails are usually significant of trophic changes in the nail; less commonly they are due to injury of the matrix by picking at the base of the nail.

Capillary Pulse.—The capillary pulse will be described in the section on the examination of the circulatory organs; it is a valuable sign of aortic regurgitation.

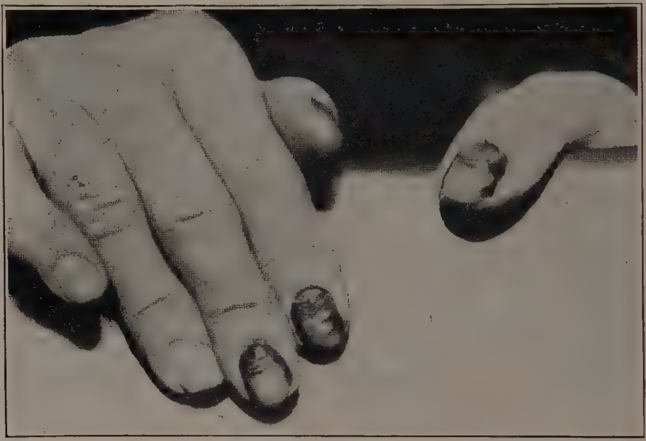


Fig. 48.—Hypertrophy of the nails. (From Hazen.)

Transverse Groove.—A transverse groove on the back of a nail is a sign of a recent acute illness. The groove has its inception at the base of the nail and its distance from the base when observed may indicate when convalescence from the illness in question commenced. Thus it requires six months for the groove to progress from the base to the free edge of the nail; hence, if it be encountered half way between the matrix and the free edge, it is an indication that convalescence began approximately three months previously.

Longitudinal ridges in the nails are said to be a reliable sign of gouty diathesis. Certainly the ridges are encountered in many gouty patients.

Incurvation of the nails, with or without clubbing of the finger tips is a sign of chronic disease of the heart or pulmonary tissues, such as cardiac insufficiency, aneurysm, phthisis, or emphysema. The incurvation may be lateral or longitudinal, or may occur in both directions.

Hypertrophy of the nails, particularly in the transverse direction associated with thickening and sometimes with twisting, occurs after acute fevers, particularly following typhoid fever, in connection with syphilis, and in sclerodactyly. A similar hypertrophy of the nail may result from eczema, may be encountered in a subject with Raynaud's disease, and in pulmonary osteoarthropathy. The nail may be simply hypertrophied without any defect in its structure (megalonychosis); or in addition to hypertrophy the nail may be twisted spirally (onychogryposis).

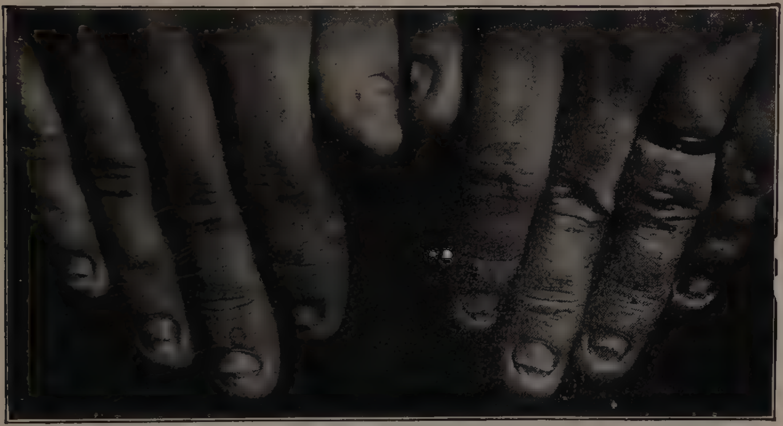


Fig. 49.—Symmetrical atrophy of nails. (Courtesy of Dr. J. C. E. King and Dr. H. G. Parker.) (From Sutton.)

Atrophy of the nails, with ulceration at the base, occurs in Morvan's disease, a syndrome which develops as a sequence of neuritis and syringomyelia. Atrophy of the nail may follow psoriasis of the fingers.

Arrested Growth.—The growth of the nails is impaired or ceases on the paralyzed side in hemiplegia. A similar arrest of growth of the nails of the paralyzed limb occurs in infantile paralysis. Arrest of growth of a nail may be detected by staining the nails at identical points upon the two hands and observing any discrepancy between the growth of the nails.

Excessive brittleness of the nails is noted in persons of gouty diathesis, the nails frequently presenting the longitudinal striations which have been described.

Onychia, ulceration of the nail matrix, occurs in children with hereditary syphilis, or scrofula, and it is said, in persons who are addicted to the chloral habit.

Paronychia, or whitlow, an acute inflammation of the tissues surrounding the matrix of the nail, may be a sequence of local trauma or may be caused by lateral hypertrophy of the nail.

Indolent Sore.—An indolent sore near the root of the nail, if indurated and associated with enlargement of the epitrochlear lymph glands, is usually a chancre; but may be due to tuberculosis.

The Fingers

The size of the finger tips in relation to the remainder of the finger may vary in healthy individuals, the greatest care being necessary at times to differentiate the normal from the abnormal. All local prominences or depressions and joint enlargements, with or without impaired joint movement, should be noted.

Tophi.—Tophi are concretions of sodium biurate which occur in the joints of the fingers in gouty subjects. They are more prominent on the dorsal surface of the joints, and may break through the skin, when they constitute the "chalk stones" of the disease.

Enlarged Joints.—Enlargement of the joints of the fingers is seen in connection with gout and rheumatism. In rheumatic fever the enlarged joints are often hot and painful.

Heberden's Nodes.—These nodes, also termed Haygarth's Nodosities, are knobby enlargements of the proximal ends of the terminal phalanges. They are noted in gout and in arthritis deformans, in which diseases they are said to be of good prognostic significance.

Clubbed Fingers (Hippocratic Fingers).—Clubbing of the terminal phalanges accompanies many chronic diseases of the heart and lungs, notably chronic bronchitis, emphysema, phthisis, chronic pleurisy, and uncompensated cardiac disease. The nails are commonly incurved. An exaggeration of this condition with swelling of the carpal joints is noted in pulmonary osteoarthropathy.

Distortions of the fingers accompany gout, chronic arthritis, and arthritis deformans. The distortions are not produced merely by fixation of the fingers in abnormal positions, but are produced by organic changes, in gout by the deposition of sodium biurate in the joints, and in arthritis deformans by absorption of bone and the growth of exostoses. The fingers are most frequently deflected toward the ulnar side of the hand.

Dactylitis.—Dactylitis is usually a sign of hereditary syphilis, and less frequently of tuberculosis. In the evolution of the deformity a fusiform purple swelling, which is prone to undergo ulceration with sinus formation, appears upon one or more of the fingers, most frequently involving the proximal phalanges.

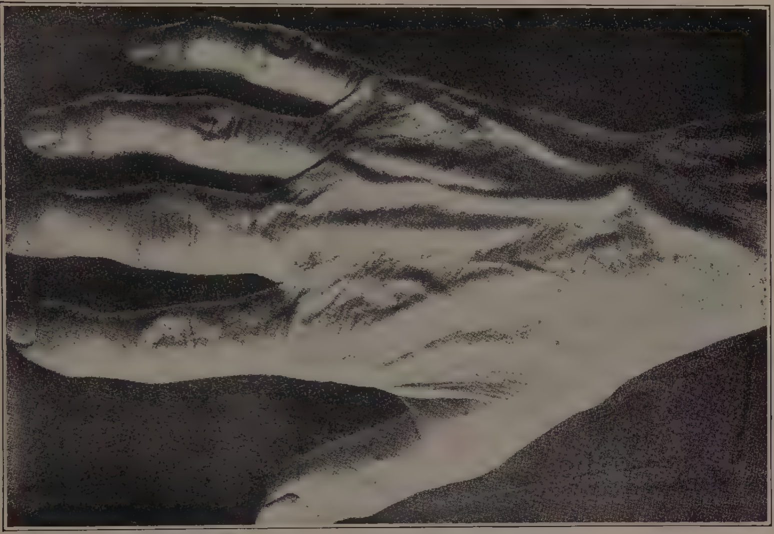


Fig. 50.—Heberden's nodes. (From Butler.)

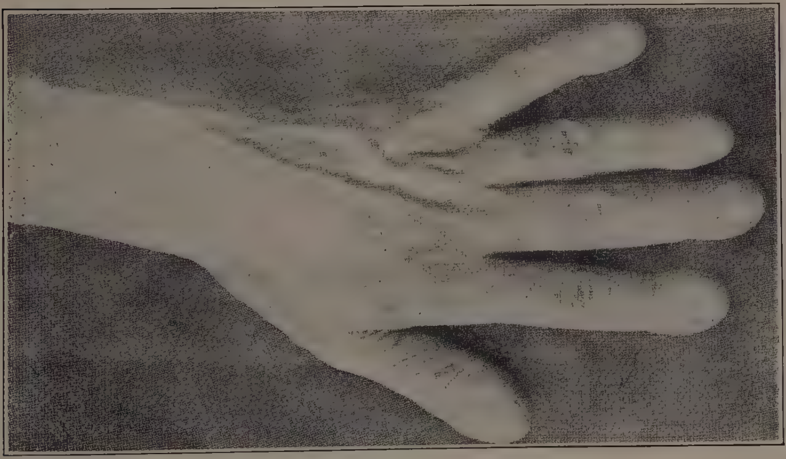


Fig. 51.—Clubbed fingers. (From Butler.)

Raynaud's Disease.—In Raynaud's disease, or "dead fingers" the fingers are bluish-black or livid, gangrene occurring in spots and leading in many instances to spontaneous amputation of the fingers.

Morvan's Disease.—In this disease the fingers are the site of painless, destructive whitlows, which have their inception adjacent to the base of the nail, leading to necrosis of the terminal phalanges and marked swelling of the fingers. The change is one of the manifestations of the syndrome of syringomyelia.

THE FOREARMS

The forearms should be compared as to size, as well as muscular development and coordinated movement. The condition of the skin should be observed as to scars, eruptions, etc.

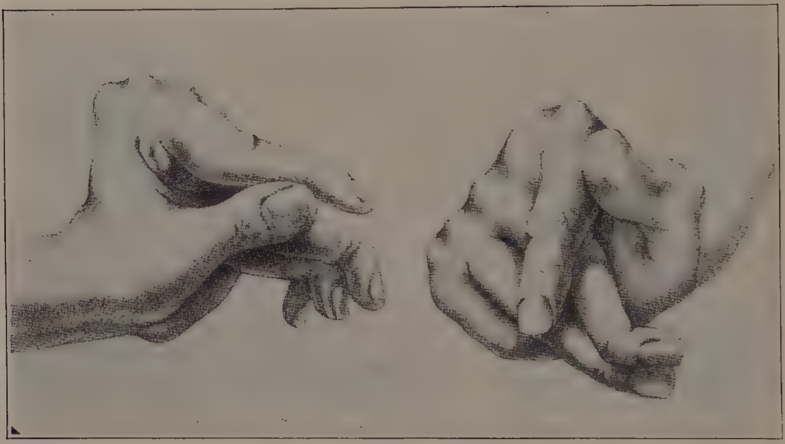


Fig. 52.—Arthritis deformans. (From Butler.)

The blood vessels vary a great deal both in size and position. This is especially true of veins. It is not uncommon to receive a report that a patient has no superficial veins from which to obtain blood for examination, only to find that only one arm has been examined.

Paralysis of the muscles of the forearm may be complete or partial, and result from injury or disease of the peripheral nerves or of the central nervous system (Klumpke paralysis).

Epiphyseal enlargement of the forearm bones at the wrist is indicative of rickets.

Enlargement of the lower end of the radius with clubbing of the fingers occurs in pulmonary osteoarthropathy, the result of chronic pulmonary or cardiac disease.

Fracture of the lower end of the radius (Colles' fracture) gives rise to a peculiar deformity of the wrist known as "silver fork deformity." Enlargement of the wrist may occur in acute or chronic arthritis.

Inspection of the region around the elbow joint may show evidence of old fractures with displacement of the olecranon.

Miner's Elbow.—In this condition there is a swelling overlying the olecranon bursa, produced by chronic bursitis of this structure, which sometimes yields fluctuation on palpation.

Enlargements or nodes along the shaft of the radius or ulna are usually due to syphilitic periostitis.

Erythema nodosum occasionally occurs upon the forearm, manifesting itself by the appearance of elevated, red, shining nodular swellings, which are very painful upon pressure.

Edema of the forearm, usually affecting the arm as well, results from thrombosis of the axillary vein, or from the pressure of mediastinal tumors upon the subclavian vein.

Bilateral edema occurs in decompensated heart disease and certain types of nephritis.

THE ARMS

Inspection of the arms should be conducted in the same manner as that of the forearms. Movements at the shoulder joint should always be compared with those of the opposite side.

Tumors.—A superficial tumor arising in the arm is most apt to be lipomatous. It is often lobulated. A ruptured biceps produces a tumor over the lower portion of the arm which increases in size when an attempt is made to flex the forearm. A deeply seated tumor of the arm is likely to prove to be a sarcoma of the humerus. An acute painful swelling of the humerus following typhoid fever or scarlatina is due to acute periostitis.

Small nodular elevations upon the humerus are usually the result of syphilitic periostitis.

Paralysis.—Paralysis of one arm may be total, the arm hanging limply, without power of movement, or may be partial. Partial brachial paralysis may assume one of two types; namely, the upper arm type of Duchenne-Erb, or the lower arm type of Klumpke. Brachial paralysis may be the result of trauma at birth, or may result from compression of the brachial plexus by a tumor or by a crutch. Some cases are due to a faulty position of the arm during anesthesia. The upper arm type of Duchenne-

Erb involves the deltoid, brachialis anticus, triceps, supinator longus, supinator brevis, and the infraspinatus muscles. The patient is unable to adduct the arm, and the forearm remains in a position of extension and pronation.

Rigidity and Contracture.—In hemiplegia the paralysis of the arm is spastic and is followed in the course of time by permanent contracture. The elbow is maintained in a state of semiflexion, the wrist is flexed upon the forearm, while the arm is often closely apposed to the trunk. Spastic rigidity of the arm is often one of the earliest signs of chronic hydrocephalus.

Movements.—In Sydenham's chorea the arms participate in the purposeless movements of the head and face. In this form of chorea there is no motor weakness; whereas in the so-called *hemiparalytic chorea,* which is attended by similar purposeless movements of the arms, the muscular power is usually impaired. Pregnant women occasionally exhibit similar purposeless movements of the arms in the so-called *chorea gravidarum.*

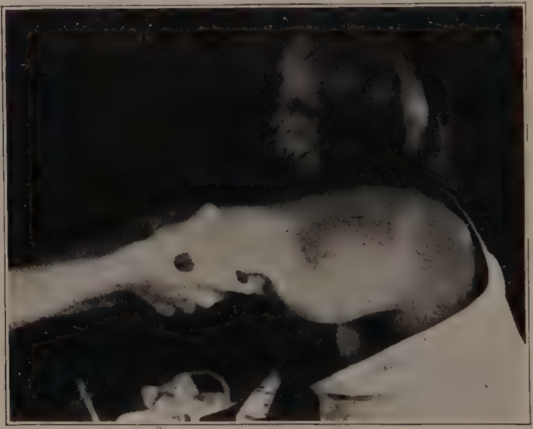


Fig. 53.—Lipoma of arm.

In *paramyoclonus multiplex* there is frequently noted a symmetrical, bilateral, clonic spasm of the muscles of the arms. The biceps, triceps, and deltoid muscles are involved. The paroxysm is characterized by a series of very rapid clonic contractions of symmetric groups of muscles in the two arms, the contractions often exceeding a hundred in a minute. Usually of very brief duration, the paroxysm may in some cases last for several moments.

Atrophy.—Atrophy of the muscles of the arm follows the paralysis of acute anterior poliomyelitis and brachial palsies, conditions which involve the lower motor neurone.

CHAPTER XII

INSPECTION OF THE THORAX

During inspection of the thorax the clothing should be removed to the waist. A light drape or kimono may occasionally be found necessary in examining patients of the opposite sex, but it is likely to cause annoyance by producing abnormal shadows and distract the examiner by falling over the field of vision.

General inspection of the thorax should be made whenever possible in the erect posture, but certain observations require the recumbent position. The subject should sit before the examiner on a revolving stool, the height of which can be adjusted, as with a piano stool; thus, the subject can be viewed from the front, back, and side without disturbing the position of the examiner or the relation to the source of light. The subject should be relaxed and comfortable. (A cold room may produce shivering or muscular contractions which interfere markedly with respiratory movements.)

During inspection of the front and back the hands of the subject should hang at the sides, but during the examination of the lateral regions they should be clasped behind the head to facilitate free inspection of the axillary regions.

The examiner should sit before the subject in such a position that his eyes are about on a level with the subject's shoulders. Later, he may stand behind the subject, looking down upon the thorax.

When examining the subject in the recumbent position the examiner stands first at the foot, and then at the head, with light falling over his shoulder obliquely upon the portion of the body to be examined.

Daylight is preferable to artificial light, and diffused light to direct sunlight. Where movements of the thorax are to be studied both direct and oblique illumination should be used if possible.

THE NORMAL THORAX

The size and shape of the thorax varies in normal subjects, and the various modifications to which they are subject can be recognized only after extensive clinical experience.

Two general types of normal thorax may be distinguished; namely, the *inspiratory* or *sthenic* type of the physically robust subject; and the *expiratory* or *asthenic* type of the subject of moderate physical development. In the latter the thorax is rather shallow with moderate anteroposterior flattening of the infraclavicular and mammary regions, slightly elongated, and presents a rather acute subcostal angle; whereas in the former the chest is deep, the anteroposterior diameter of the thoracic cavity approaching but not attaining to the dimensions of the lateral diameter, and with quite an obtuse subcostal angle.

In the normal subject the shoulders usually occupy the same level, though moderate dropping of one shoulder is not infrequent. The clavicles are not unduly prominent; but moderate depression of the supraclavicular and infraclavicular fossæ is not incompatible with perfect physical well being. The two halves of the thorax are seldom perfectly symmetrical, as the right side is usually more fully developed than is the left side owing to the greater employment of the right arm in right-handed individuals.

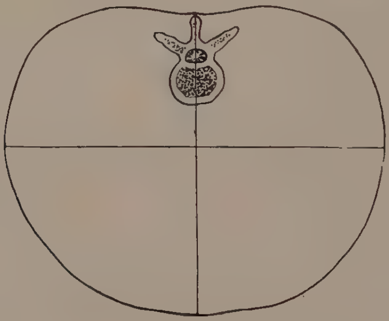


Fig. 54.—Cross section of normal thorax.

The sternum of the normal thorax presents a moderate forward convexity, which attains its maximum degree in the central portion of the bone. At the junction of the manubrium with the gladiolus the sternum usually is marked by a transverse ridge, the angle of Louis, which corresponds to the level of the second chondrosternal articulation. The ensiform cartilage may be depressed or may exhibit a moderate forward inclination without possessing untoward significance.

In the inspiratory or asthenic type of thorax there is frequently a moderate anteroposterior convexity of the thoracic wall at the junction of the ribs with their costal cartilages, whereas in the thorax of the expiratory or asthenic type there is apt to be flattening at the costochondral articulations over the upper portion of the thorax.

The normal bony thorax is clothed with a muscular and subcutaneous tunic of moderate thickness so that the intercostal spaces are neither depressed nor unduly prominent. The thoracic skin is smooth and is lubricated by a moderate amount of sebaceous material.

Upon cross-section the normal thorax is reniform, with the hilum which corresponds to the vertebral column directed posteriorly, the transverse diameter exceeding the anteroposterior diameter by one-fourth in the adult subject. In the thorax of the child this relation between the transverse and the anteroposterior diameters of the cavity does not obtain, as the infantile thorax is almost circular upon section. In the aged the anteroposterior diameter again becomes greater, due to pulmonary emphysema and ossification of the costal cartilage.

THE CHEST WALL

Inspection of the chest wall may reveal changes in the condition of the skin, alterations in the subcutaneous tissues and musculature, changes in the superficial vessels of the thorax, changes in the mammary gland, or abnormalities of the ribs and intercostal spaces.

The *skin* of the thorax in health is smooth and glossy and is lubricated with an adequate amount of sebaceous material. The skin of the adult male thorax is frequently clothed with a variable amount of hair, which is apt to obscure the true state of the skin and which may become a source of error in the application of the various maneuvers which are employed in the physical examination of the thorax.

The *color* of the skin varies normally in different individuals. Aside from this normal variation in pigmentation, certain diseases which are not directly referable to the respiratory organs produce more or less characteristic pigmentation of the skin, notably Addison's disease, syphilis, chronic malarial intoxication, hepatic disorders, and following herpes zoster; whereas chlorosis and pernicious anemia lend a greenish or lemon-yellow tint to the skin of the thorax as indeed to that of the entire body. In arthritis of long standing the thoracic integument is shiny and frequently covered with the pustules of acne. In the cachexia of malignant disease it is thinned and presents a yellow, straw-colored tint; while in advanced chronic phthisis the skin of the thorax is dry, with general pallor, and with here and there irregular, yellowish spots of pityriasis versicolor from which it is easy to detach a thin layer of epidermis. The skin of the thorax is subject to the eruptions of the acute, exanthematous fevers; and the skin of this region not infrequently presents scars, the result of trauma or of syphilis.

Occasionally the examiner will encounter silvery striæ, analagous to the striæ developing upon the abdomen during pregnancy in patients who have suffered from thoracic distention during early life and adolescence. The striæ are ordinarily situated upon the posterior and inferior portions of the thorax and pursue a course parallel with the intercostal spaces. They are observed in the train of extensive lobar pneumonia, serofibrinous pleurisy with effusion, chronic ulcerative phthisis, and pneumothorax. In these pathologic states of the thoracic viscera the striæ develop upon the side opposite the disease process which cripples the corresponding lung and leads to vicarious distention of the opposite lung as a consequence of the compensatory emphysema which develops. Gilbert, Troisier, and Menetrier explain their production as the result of rupture of the elastic fibers of the deeper portions of the thoracic integument along the course of the intercostal spaces, a rupture which is especially likely to occur in this class of patients on account of the unusual elasticity and resilience of the thoracic cage at this time of life.

The state of the *subcutaneous tissues* and *musculature* of the thorax is influenced by the general nutrition of the patient, and varies as this is good, moderately good, or poor. Local degeneration and atrophy of these tissues has been shown by Pottenger to be indicative of old and probably quiescent pulmonary lesions, whereas a spasm of the musculature attends active disease of the lung or pleura. While these changes are frequently to be detected upon inspection of the thorax, light palpation with the fingertips is more serviceable in demonstrating the condition.

The *superficial veins* of the thoracic wall, scarcely visible in the normal subject, become engorged and tortuous when intrathoracic lesions interfere with the venous return to the right heart. Thus, engorgement of the superficial veins of the thorax is significant of compression of the large intrathoracic venous trunks by mediastinal tumor, aortic aneurysm, or excessively hypertrophied heart. Similarly, distention of the veins over the lower region of the thorax, communicating with similarly distended veins over the abdomen, and not infrequently associated with the caput medusæ, is indicative of stasis in the distribution of the portal vein and inferior vena cava.

The *mammary gland,* an imperfectly developed and rudimentary structure in the male subject, in the presence of pulmonary tuberculosis in this sex has occasionally been observed to undergo an extensive hypertrophy. In other instances hypertrophy of this gland in the male sex is congenital and devoid of diagnostic significance. In the latter event the glandular hypertrophy is commonly bilateral. In the female subject, on the contrary, hypertrophy of the mammary gland is a normal accompaniment of pregnancy and lactation, and enlargement of the gland also attends malignant disease.

Edema of the thoracic wall may be encountered in a general or a local form. General edema of the chest wall attends general anasarca. Moderate edema of the thoracic wall, limited to one side of the thorax, is almost invariably indicative of suppurative disease within the thoracic cavity. When it develops in the course of serofibrinous pleurisy with effusion, it is suggestive of a purulent alteration of an effusion which has been serofibrinous. When empyema is rapidly extending and rupture through the thoracic wall is imminent, there commonly develops a localized, edematous prominence, usually with moderate discoloration of the integument, *empyema necessitatis.* When this is located upon the left side of the thorax, in close proximity to the heart, the contractions of the heart are apt to provoke systolic pulsation of the contained purulent material, with the consequent production of a condition of *pulsating empyema.*

The *intercostal spaces* are normally slightly depressed, contrasting with the adjacent ribs. In the presence of wasting of the subcutaneous tissues and intercostal musculature as the result of the emaciation of chronic pulmonary tuberculosis, diabetes mellitus, or paralysis of the intercostal muscles, the normal depression of the intercostal spaces is accentuated, rendering the ribs unduly prominent. On the other hand, in the presence of extensive pleural effusion and in pneumothorax the intercostal spaces upon the affected side occupy the same plane as their corresponding ribs, in marked contrast to the normal depression of these spaces upon the opposite side of the thorax. Actual bulging of the intercostal spaces is very rarely encountered, save in empyema of extensive duration.

The *ribs,* not clearly perceptible upon inspection in the normal subject save in the lower axillary and infraaxillary regions, in the presence of chronic wasting disease become conspicuous landmarks upon the surface of the thorax. In rickets, moreover, there is permanent deformity at the junction of the ribs and costal cartilages, constituting the *rachitic rosary* of this disease.

The course pursued by the ribs is suggestive of certain diseases of the respiratory organs. In the alar thorax of chronic ulcerative phthisis the ribs pass sharply downward from the vertebral column, bending sharply upward to meet the sternum, resulting in a very acute subcostal angle. In the barrel chest of hypertrophic emphysema, on the contrary, the ribs pass horizontally forward from the vertebral column with the consequent production of a very obtuse subcostal angle. In rickets the ribs are compressed laterally, with consequent diminution in the lateral diameter of the thorax, resulting in the keel or pigeon breast.

The *costal cartilages,* normally elastic and mobile, with advancing age become progressively less resilient through advancing calcification, limiting the freedom of excursion of the thorax during respiration.

The axilla is smooth in the child, but at puberty a varying growth of hair appears. The sebaceous glands are numerous, as are also sweat glands, so that in many individuals the axilla is constantly moist and during excitement drops of perspiration course down the thorax. There are numerous lymph glands in the axilla which become enlarged following infection of the upper extremity. They are also apt to take up metastatic cells from carcinoma of the breast.

Primary abscesses in the axilla are very common in children and young adults. They may be followed by considerable scarring and permanently enlarged lymph nodes.

Examination of the chest wall should include the back. The skin in general resembles that of the front of the thorax, but hairy growth is less common and less abundant. Sebaceous glands are numerous, and acne pustules are very common, especially during adolescence.

The scapulæ in the subject of the sthenic type lie close to the ribs and move smoothly as the shoulders are thrown forward. The scapulæ in the asthenic type are less firmly placed and tend to pull away from the thorax when the shoulders are thrown forward. They are sometimes spoken of as "winged scapulæ."

Positions and movements of the scapulæ should be observed as the subject sits erect, throws his shoulders forward and extends the arms. The vertebral borders of the scapulæ are normally curved outward, but in congenital syphilis they are often concave (scaphoid scapulæ).

The spinal column in health curves slightly backward from the seventh cervical to the fifth dorsal vertebra, then curves forward to the second lumbar, and again backward in the sacral region. The spines of the vertebræ should be carefully observed for alignment.

A pronounced backward curve of the vertebra is known as kyphosis; a pronounced forward curve, lordosis; a pronounced lateral curve, scoliosis. Such curvatures are usually the result of tuberculosis or rickets, involving the bodies of the spinal vertebræ. The subject should be instructed to bend forward, backward, and to the right and left. Any irregularity of move-

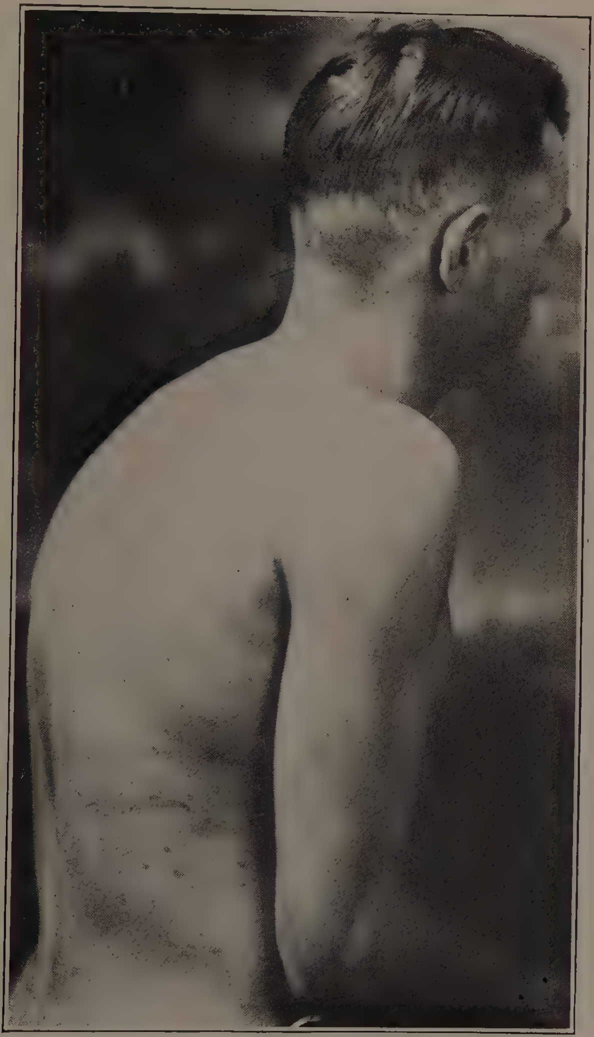


Fig. 55.—Kyphosis due to vertebral caries.

ment or stiffness should be noted, as well as any weakness or paralysis of back muscles.

Tumors of the spine may be due to spina bifida, tuberculosis, sarcoma, or metastatic hypernephroma. Other tumors on the back are usually fatty or due to intrathoracic disease.

Pulsations may be observed in the left back, due to aneurysm of the descending portion of the aorta. A retraction of the chest wall, synchronous with the systole of the heart, is noted at times when adhesions occur between the pericardium and diaphragm or pleura (Broadbent's sign).

DEFORMITIES OF THE THORAX

Deformities of the thorax may be congenital; they may be the result of occupation; or they may be caused by pulmonary or by constitutional disease. In the main, deformities of the thorax, if not due to congenital maldevelopment or to rickets, are caused by obstruction of the upper respiratory passages from hypertrophied tonsils or adenoid vegetations, in children; while deviations from the normal contour of the thorax in the adult are caused by, and are secondary to, pathologic changes in the thoracic viscera, such as collapse or fibroid retraction of a lung, excessive inflation of the lungs by hypertrophic emphysema, the presence of aneurysm of the thoracic aorta, excessive cardiac hypertrophy, or the development of an intrathoracic neoplasm. From these considerations, it follows that deformity of the thorax may be bilateral, unilateral, or local, depending upon the nature of the underlying etiologic factors.

Bilateral Deformities

The Emphysematous Thorax (Barrel-chest).—In hypertrophic emphysema the thorax presents characteristic deviations from the normal contour, constituting the barrel-chest of this disease. The thorax is increased in all of its diameters, but particularly in the anteroposterior diameter, which in marked contrast to the normal thorax, exceeds the transverse diameter. The thorax is thick and relatively short, and is maintained in a position of distention which exceeds that which normally obtains at the completion of full inspiration. The thorax upon cross section is almost circular, with the maximum degree of enlargement corresponding to the level of the central point of the sternum.

The thorax presents the greatest degree of distention in the upper and central portions, for the reason that the chronic catarrhal inflammation of the smaller bronchioles throughout the voluminous bases presents an obstacle to the free ingress of the inspired air to these portions of the lungs, whereas the apices and anterior borders of the lungs expand compensatorily. For the same reason, the excursion of the diaphragm during respiration is limited, throwing an increased burden upon the upper intercostal muscles and the accessory muscles of respiration, all of which tends to accentuate the

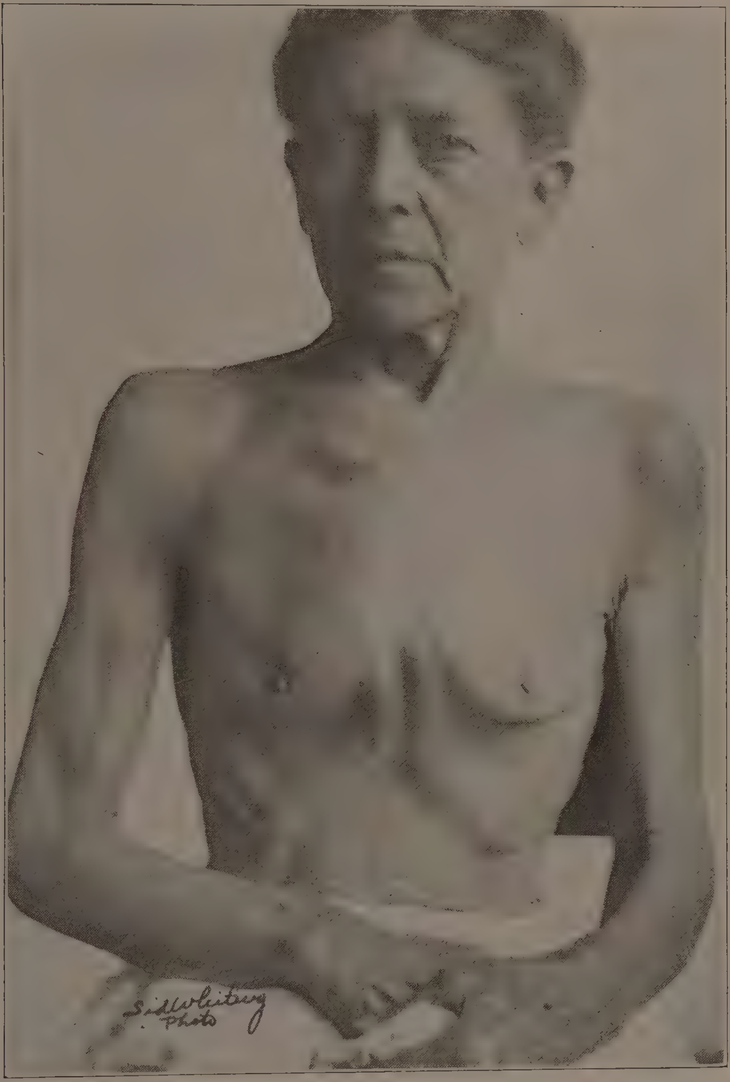


Fig. 56.—Emphysemic chest. (Front view.)

discrepancy between the expansion of the upper and lower halves of the thorax. However, owing to the acquired rigidity of the costal cartilages in the emphysematous thorax, there is little expansion of the chest; but in compensation for this deficiency on the part of the normally resilient costal cartilages, the thorax during respiration rises and falls *en masse.*

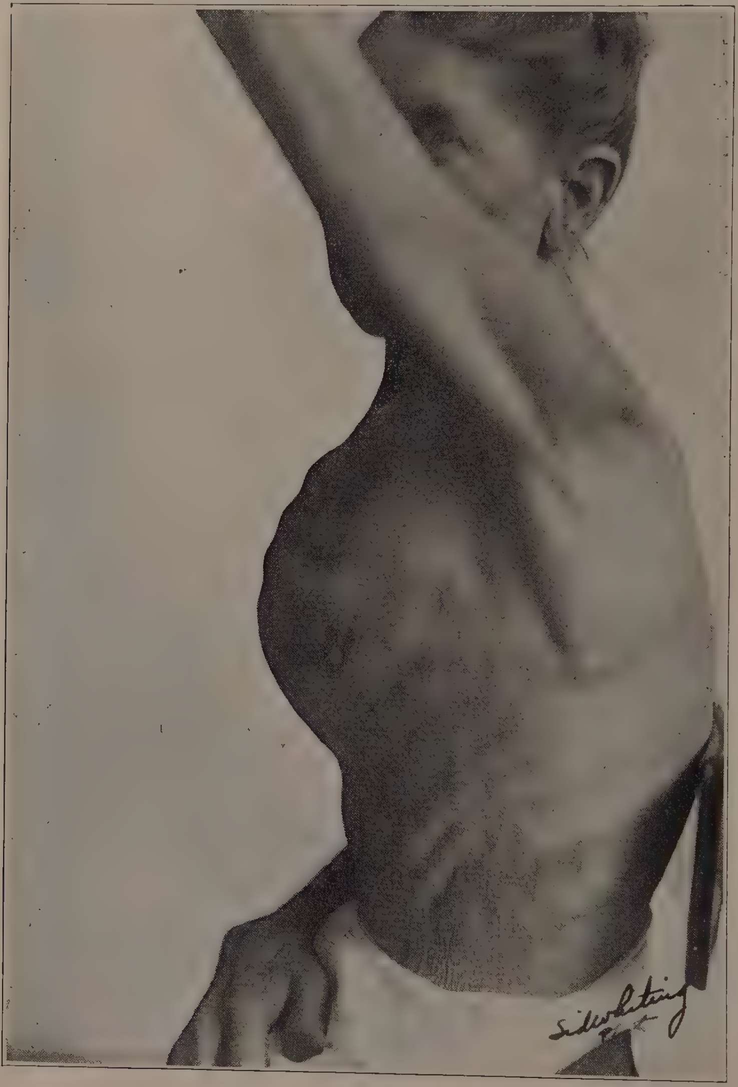


Fig. 57.—Emphysemic chest. (Lateral view.)

The ribs in the upper half of the thorax pass almost horizontally forward from the vertebral column to meet the sternum, while the normal obliquity of the lower ribs is likewise diminished, resulting in a very obtuse costal angle, and a corresponding increase in the outlet of the thorax. Owing to the horizontal course of the ribs and the general distention of the thorax, the intercostal spaces are abnormally wide, and peculiarly hard and unyielding upon palpation. Not infrequently there is visible retraction of the lower intercostal spaces during inspiration.

As a rule, the sternum occupies the same plane as the costal cartilages, without noticeable bulging or recession, though the bone is not infrequently bent slightly at the junction of the manubrium and gladiolus, resulting in an unduly prominent angulus Ludovici.

The scapulæ are closely applied to the back of the bony thorax. The normal dorsal curve of the vertebral column is accentuated, simulating kyphosis in the extreme case. The clavicles are elevated, and the neck is short and thick, often with conspicuous engorgement of the cervical veins. The sternomastoids are unduly prominent and the head is slightly thrown backward in the effort to bring the accessory muscles of respiration into play.

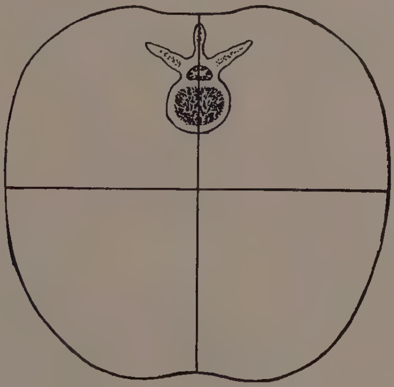


Fig. 58.—Cross section of emphysematous thorax.

The condition of the supraclavicular and infraclavicular fossæ is variable. Frequently these spaces have disappeared and the integument occupies the level of the clavicle or even presents a local bulging.

Kyphosis acquired during laborious occupations or developing as the result of dorsal caries may be mistaken for the barrel-chest of emphysema, as may also thoracic distention due to malignant disease of the thoracic viscera, or thoracic deformity due to extensive pleural effusion or to enlargement of the liver or of the spleen.

The Phthisical Thorax (Alar, Pterygoid, or Paralytic Thorax).—The phthisical thorax presents a marked contrast to the emphysematous chest. In the phthisical thorax the anteroposterior diameter is greatly diminished, and the thorax is long and flat, presenting a picture of extreme emaciation, owing largely to wasting of the pectoral and deltoid muscles. The clavicles stand out prominently, in striking contrast to the deep recession of the supraclavicular and infraclavicular fossæ above and below the bones.

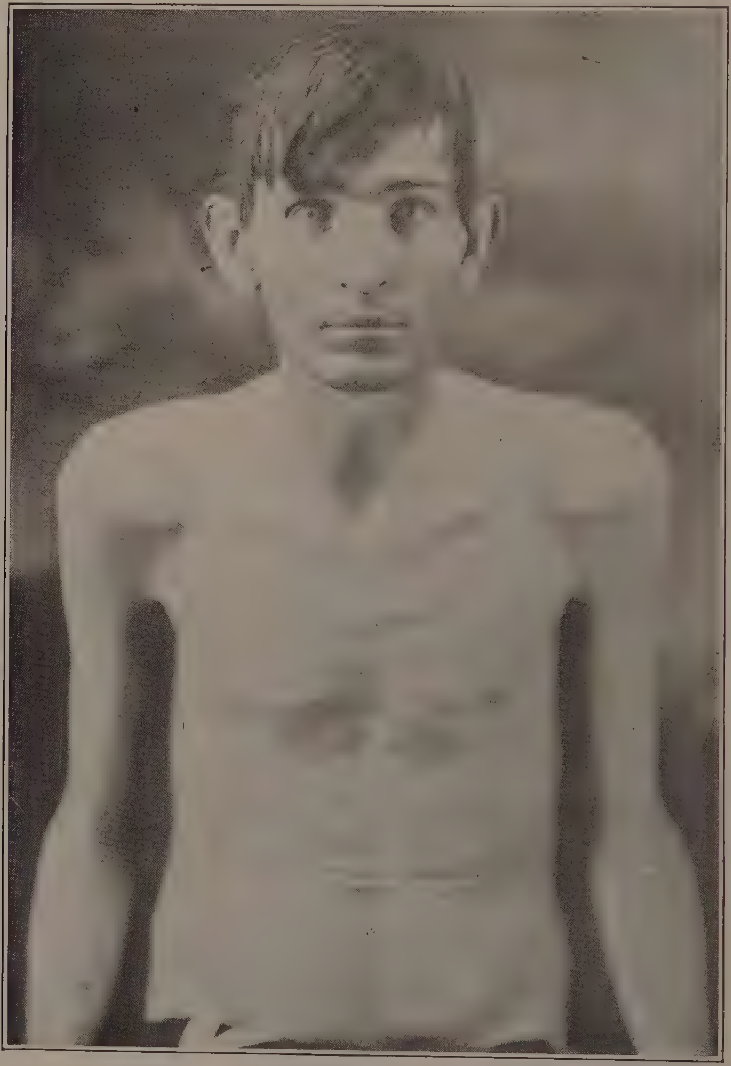


Fig. 59.—Phthisical thorax. (Anterior view.)

The inner borders of the scapulæ stand out upon the posterior thoracic wall like wings, hence the name "alar thorax" which is frequently applied to this type of thoracic deformity. The wing-like scapulæ enjoy an abnormal mobility upon the posterior thoracic wall, owing to atrophy of the investing musculature. The shoulders, which seldom occupy the same level, are inclined forward, while the head habitually droops anteriorly.

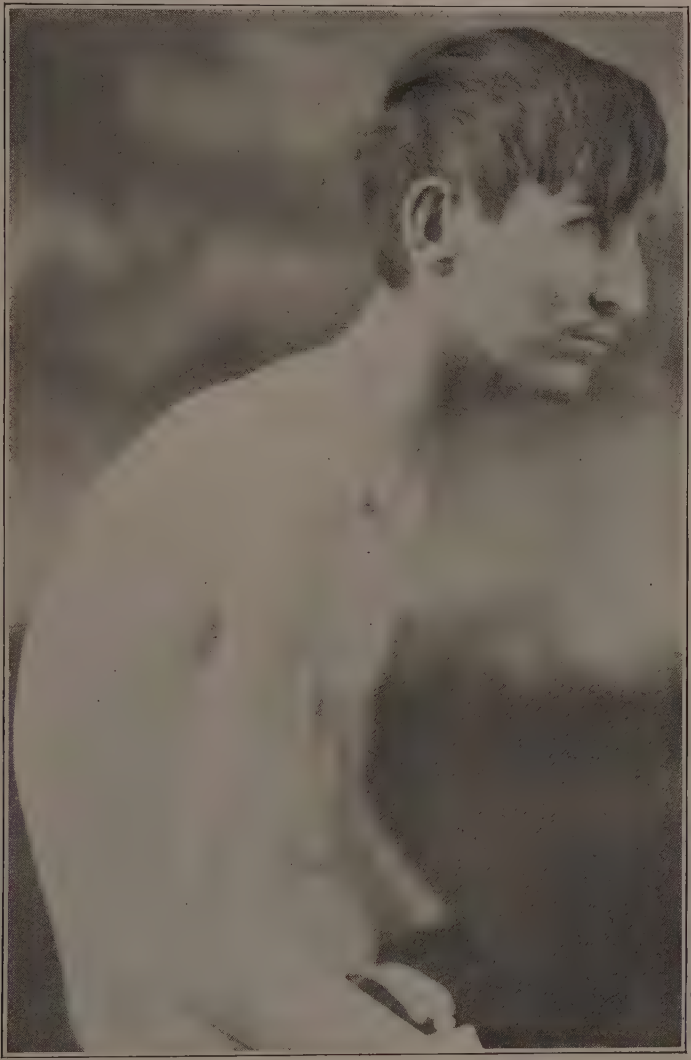


Fig. 60.—Phthisical thorax. (Lateral view.)

The ribs pursue a very oblique course downward from the vertebral column and bend sharply upward in front of their angles to meet the sternum, producing a very acute subcostal angle. The intercostal spaces are wide; and the intercostal muscles are wasted, yielding readily to palpation with the finger-tips. The elongated, flat thorax is surmounted by a long, tapering neck in which the sternomastoid muscles and the larynx stand out prominently.

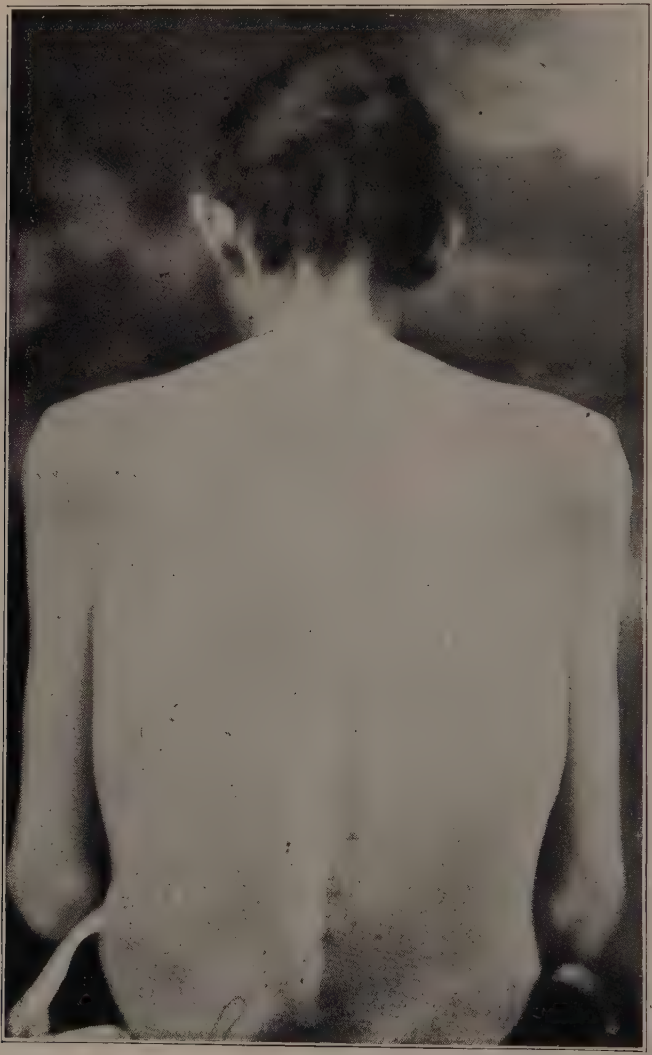


Fig. 61.—Phthisical thorax. (Posterior view.)

The phthisical thorax in its fully developed form is encountered in certain cases of chronic phthisis, but it is by no means uniformly present, and a diagnosis of phthisis may not be made upon the configuration of the thorax alone. In its partially evolved form the thorax is encountered in certain cases of inanition, and it occurs as a manifestation empyema of extensive duration. It has been suggested that this type of thoracic deformity may be caused by tuberculosis of the bronchial glands arising during childhood.

The Rachitic Thorax.—Advanced rickets is accompanied by a characteristic deformity of the thorax. In this type of thoracic deformity the anteroposterior diameter is increased, while the lateral diameter is diminished as the result of muscular action upon the abnormally yielding ribs, causing the sternum to jut forward and to assume an unduly prominent position. A cross section of the rachitic chest shows a marked increase in the anteroposterior diameter with an actual decrease in the transverse diameter of the thorax.

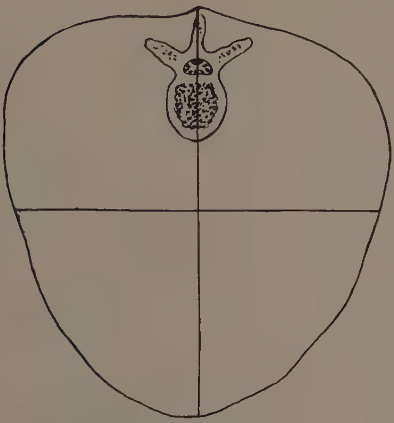


Fig. 62.—Cross section of rachitic thorax.

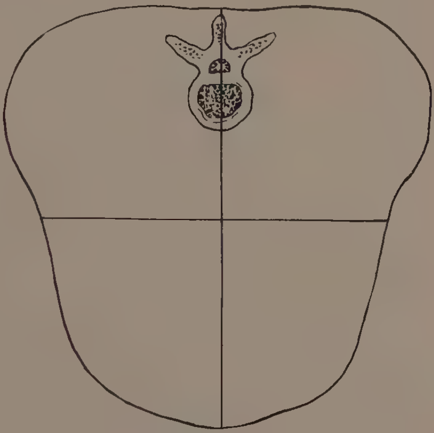


Fig. 63.—Cross section of pigeon breast.

At the junction of the ribs with their costal cartilages the rachitic thorax presents a series of nodular swellings, due to enlargement of the osteocartilaginous junctional tissues, the *rachitic rosary*. As a result of the lateral compression of the thorax, the costal angle is abnormally acute. Not uncommonly the lower ribs flare outward anteriorly. The rachitic thorax is attended by various types of spinal curvature, as kyphosis, lordosis, or scoliosis.

The rachitic chest is not significant of any disease of the respiratory organs; but the compressed thorax is not sufficiently capacious for the lungs to properly expand and to attain their full development, and hence this type of thoracic deformity predisposes to disease of the organs of respiration.

Harrison's Sulcus.—Harrison's sulcus is a groove or depression extending downward and outward upon either side of the thorax from the ensiform cartilage toward the infraaxillary regions. It is often a sign of early rickets; it almost invariably accompanies the rachitic thorax; and it is also caused in early life by obstructive lesions of the upper air passages, in which event it is to be attributed to the external atmospheric pressure compressing the soft ribs which are not supported by full inflation of the lungs. The deformity is particularly common in the negro race.

The Pigeon-Breast. (Keel-Breast: Pectus Carinatum).—In this type of thoracic deformity the ribs are compressed and straightened in front of their angles, causing the sternum to jut forward and to become unduly prominent. Upon cross section the thorax is roughly triangular. The anterior portion of the thoracic cavity is encroached upon by the incurvation of the ribs, while the posterior portion of the cavity is compensatorily voluminous. Harrison's sulcus is frequently present upon the lateral regions of the thorax.

The pigeon-breast occurs most frequently in cases of advanced rickets; but it may also be produced by the paroxysms of pertussis or by greatly hypertrophied tonsils during early life.

The Funnel-Chest.—In this type of thoracic deformity the lower end of the sternum is depressed, the hollow or depression occasionally extending as high as the third rib. The funnel-chest is in the vast majority of instances a congenital deformity; it has been noted in connection with advanced rickets; and it occasionally develops as the result of an occupation which requires the application of an instrument constantly against the lower portion of the sternum as in the case of the cobbler or the carpenter. The funnel-chest is not essentially a sign of pulmonary disease; but, by decreasing the capacity of the thorax, it predisposes to disease of the bronchopulmonary system.

Unilateral Deformities

Unilateral Enlargement.—Enlargement or bulging of one side of the thorax is caused by an increase in the size of the visceral content of the corresponding side, or by the presence of abnormal material, which may be air, gas, serum, pus, or blood. The enlargement may be significant of compensatory emphysema of one entire lung, arising as the result of vicarious distention of the lung to compensate for crippling of the opposite lung by cavitation, compression, or fibrosis, in which event the retraction of the diseased side of the thorax adds materially to the apparent bulging of the emphysematous side. The presence of air or gas in the pleural cavity in open or closed pneumothorax produces unilateral bulging of varying degree. The compression of the lung in these cases also results in compensatory emphysema of the opposite lung; but the latter does not attain such a degree as to equalize the enlargement of the two sides of the thorax. Extensive pleural effusion, hemothorax, empyema, and hydrothorax cause similar disproportion between the two halves of the thorax; and in these cases, in which the enlargement is due to the presence of fluid, the bulging is most pronounced in the lower portion of the thorax; the costal angle is rendered more obtuse; and the intercostal spaces occupy an equal plane with the ribs or show slight bulging.

In unilateral enlargement of the thorax the circumference and anteroposterior diameter of the affected side are increased, and the intercostal spaces are wider than normal. The shoulder upon the affected side is slightly elevated, and the vertebral column deviates toward the side of the bulging.

Unilateral Diminution.—Diminution of the size of one-half of the thorax is indicative of a corresponding diminution in the size of the intrathoracic contents upon the affected side. Chronic pulmonary tuberculosis of the corresponding lung is the most prolific cause of this deformity. In other instances it is due to bronchial obstruction with subsequent collapse of an extensive portion of the lung. Fibrosis of the lung in the course of chronic interstitial pneumonia, fibroid phthisis, and pulmonary syphilis results in unilateral retraction or flattening of one side of the thorax. A further causative factor in the production of this type of thoracic deformity is the traction of adhesions which bind the visceral to the parietal pleura, or total obliteration of the potential pleural cavity, the result of chronic adhesive pleurisy.

All of these conditions are attended by compensatory emphysema of the opposite lung, thus accentuating the disproportion between the two sides of the thorax. In this deformity the circumference of the affected side is diminished, as is the anteroposterior diameter; whereas the transverse diameter is increased. In cases due to chronic pulmonary tuberculosis, the retraction is most marked upon the upper half of the thorax; whereas in cases dependent upon chronic adhesive pleurisy the deformity is most noticeable over the lower thoracic region.

In addition to the diminution in the size of the affected side, the corresponding shoulder droops, and the vertebral column deviates, with its concavity directed toward the retracted side. This curvature of the spine is apt to prove a source of error in the casual examination of cases presenting minor degrees of unilateral retraction, as lateral spinal curvature from other causes may simulate unilateral diminution of the thorax in its absence. The intercostal spaces upon the retracted side of the thorax are narrowed; and, in cases of extreme retraction, the ribs may actually overlap in the lower thoracic region.

Local Deformities

Local deformity of the thorax may have reference to changes in the organs of respiration or to alterations in the circulatory system. Those which are referable to the former possess a variable significance, depending upon the location upon the thoracic surface in which they occur and upon their general physical characteristics.

Local Prominence.—Precordial bulging is commonly seen in children and in thin-chested people with cardiac hypertrophy or pericardial effusion. Upon the anterior or lateral thoracic wall a local prominence may indicate a localized serofibrinous pleurisy; a large neoplasm of the mediastinal structures, lung, or chest wall; or of a large pulmonary cavity which is filled with fluid. A local prominence which is associated with edema or discoloration of the integument of the thorax signifies empyema necessitatis. Rarely congenital diaphragmatic hernia is responsible for local prominence of the chest wall. Undue prominence of the sternum, when not caused by aneurysm of the aortic arch, is frequently due to malignant disease of the mediastinal glands. Local prominence of the left hypochondriac region points to splenic enlargement, while a similar enlargement over the right hypochondriac region is indicative of hepatic enlargement or of a low right-sided pleural effusion.

Local Retraction.—Undue depression of the supraclavicular and infraclavicular regions, with unduly prominent clavicles, points in the first instance to apical pulmonary tuberculosis, and secondarily to fibrosis of the lung or to traction by pleural adhesions. Local retractions upon the anterior thoracic wall, adjacent to the lateral sternal borders, frequently indicate pulmonary excavation due to chronic ulcerative phthisis or to bronchiectasis; while a circumscribed area of flattening or of moderate depression upon the posterior wall of the thorax accompanies pulmonary abscess. Local retraction over the lower portion of the sternum constitutes the essential feature of the funnel-chest, whereas upon the lateral walls of the thorax Harrison's sulcus is frequently noted in rachitic patients. As a general rule, local retractions in the lower anterior and lateral thoracic regions are the result of traction by pleural adhesions.

MOVEMENTS OF THE THORAX

A study of the movements of the thorax in health and in disease constitutes an important part of every physical examination. These movements are influenced by the rate, rhythm, and the type of respiration obtaining in a given case; and the movements are influenced and modified by disease arising within the thorax or in other portions of the body. Observation of the thoracic mobility reveals the degree of expansion of the thorax, as well as the diaphragmatic phenomenon of Litten. Finally, circumscribed mobility of the thorax is noted in the form of normal and abnormal pulsations.

Respiratory Movements of the Thorax

The respiratory movements of the thorax comprise an inspiratory excursion and an expiratory recession, the latter being followed by a slight pause during which the thorax is in a state of repose. The inspiratory movement is an active process, which is initiated and executed by muscular contraction; whereas the expiratory recession is a passive movement, the thoracic walls receding upon the cessation of the muscular action. Of the two phases, the expiratory recession is of longer duration than is the inspiratory excursion, the ratio being as 6 is to 5.

The *frequency* of the respiratory movements of the thorax in health varies with the age and sex of the subject. In the normal adult the frequency is fourteen to eighteen respirations per minute. In the newly born, on the contrary, the respiratory rate is approximately forty-four to the minute, while at five years of age it averages twenty-six respirations per minute. The respiratory rate in the female subject is normally slightly in excess of that which obtains in the male.

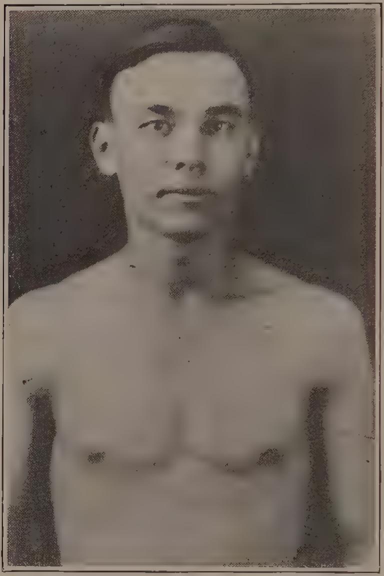


Fig. 64.—Normal thorax (in repose).

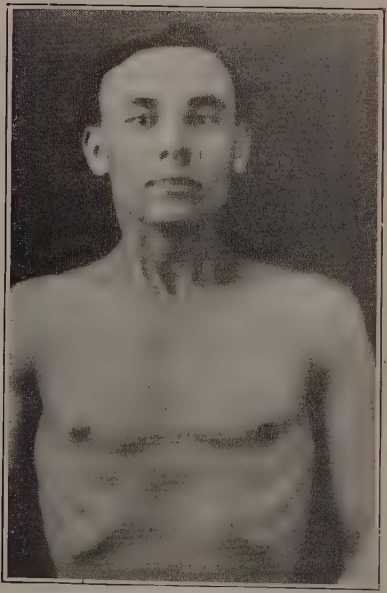


Fig. 65.—Normal thorax (full inspiration).

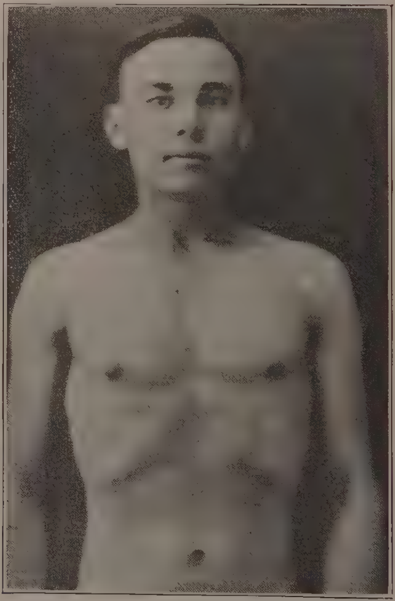


Fig. 66.—Normal thorax (full expiration).

The *character* of the respiratory movements of the thorax also varies with the age and sex of the subject, and two types of normal respiration are recognized; namely, the *costal type,* and the *costo-abdominal type* of respiration.

In the adult female subject the costal type of respiration obtains, in which the thoracic movement is much more conspicuous than is that of the abdomen. In this type of respiration the superior regions of the thorax exhibit the greatest degree of expansion, the lower thoracic walls moving less conspicuously; hence the name "upper thoracic type" which is sometimes employed to describe it. In this type of respiration the anterior

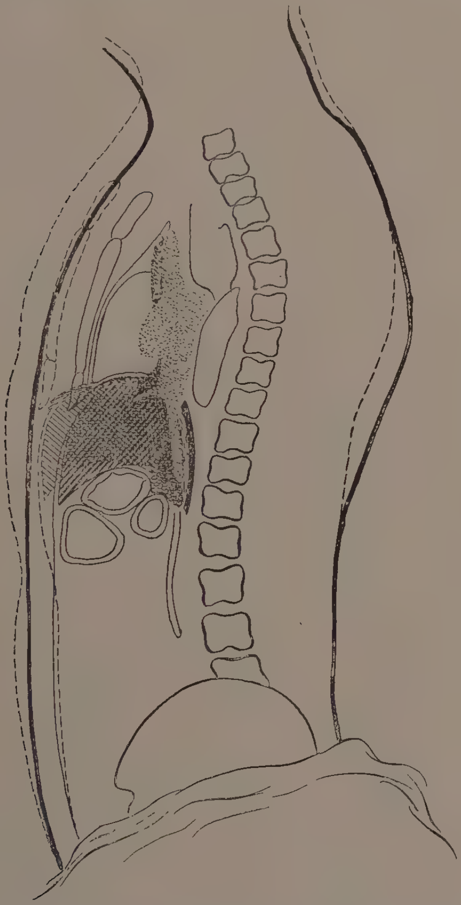


Fig. 67.—Illustrating the movements of the diaphragm and thoracic and abdominal walls, as well as the change in position of the intrathoracic and intraabdominal viscera, during respiration of the abdominal type. The movements are from the solid lines on expiration to the broken lines on inspiration. (Pottenger, after Hasse.)

and posterior chest walls are carried upward during inspiration, while the sternum is raised and thrust forward. The diaphragm participates only slightly in the inspiratory movement, so that there is little or no noticeable epigastric bulging during inspiration.

In the adult male subject and during childhood, on the contrary, the respiration is of the costoabdominal type, in which the maximum expansion is noted over the lower thorax and the upper abdomen. The diaphragm is a prime factor in the execution of this type of respiration, and by virtue of its descent during inspiration, this phase of the respiratory cycle is marked by excursion of the lower thoracic walls and epigastric bulging, while the costal

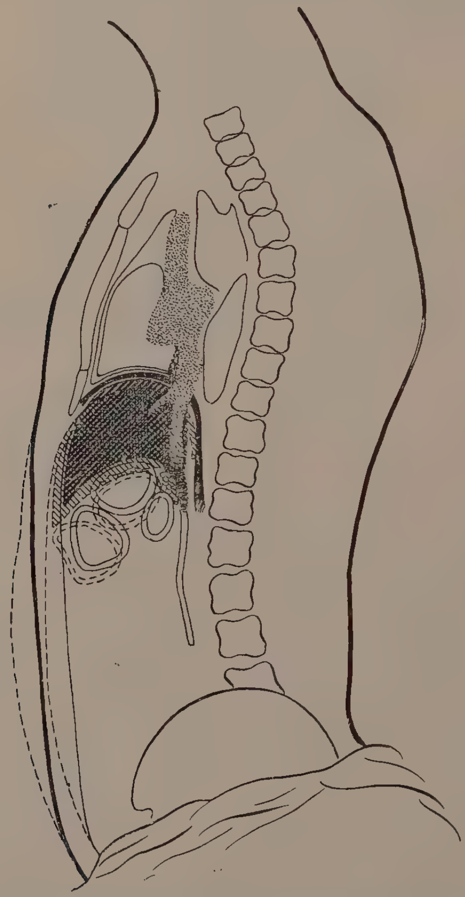


Fig. 68.—Showing the movements of the diaphragm and thoracic and abdominal walls, as well as the change in position of the intrathoracic and intra-abdominal viscera, when combined thoracic and abdominal breathing are pronounced. The movements are from the solid lines on expiration to the broken lines on inspiration. (Pottenger, after Hasse.)

angle becomes more obtuse. At the same time the sternum is elevated; the ribs assume a more horizontal position; and there is an increase in both the anteroposterior and the transverse diameters of the thorax. Women may be trained to use the costoabdominal type of respiration. The result of such training may be noted in opera singers.

Yet while these distinct types of normal respiration obtain in the two sexes, an exaggeration of either the costal or the costoabdominal type, or the substitution of the one for the other, is abnormal; and such alteration possesses definite diagnostic significance. Thus, exaggeration of the costal type of respiration in the female subject or the presence of this type in the male is indicative of immobilization of the diaphragm by subphrenic pressure or of paralysis of this important muscle. Excessive subphrenic pressure, preventing the descent of the muscle during inspiration may be due to ascites, large abdominal tumor, tympanites, or peritonitis.

Similarly, accentuation of the costoabdominal type of respiration in the male or its presence in the female subject, except as noted above, points to premature calcification of the costal cartilages or scleroderma, destroying the normal elasticity of the upper thorax; or it may signify inhibition of thoracic excursion due to the pain of an acutely inflamed pleura, of pleurodynia, or of a recent fracture of a rib. Such a reversal of the normal type of respiration is also noted in the presence of extensive disease of the thoracic viscera, notably in massive pneumonia, bilateral pleurisy with effusion, pulmonary tuberculosis, or pulmonary neoplasm.

The Diaphragmatic Phenomenon (Litten's Sign).—At the completion of expiration about one-third of the upper surface of the diaphragm is in apposition with the lateral wall of the thoracic cavity, from the seventh to the tenth ribs. At the beginning of the inspiration the diaphragm begins to descend, pulling away from the chest wall slowly and steadily during the whole of the inspiration, so that by the end of this portion of a respiratory cycle it has completely separated from the chest wall, except at its attachment. The instant the separation begins the negative pressure is increased and the lung consequently descends but does not quite keep pace with the increase of negative pressure. The result is that the thoracic wall is forced inward slightly by atmospheric pressure. A shadow-like movement usually results which is best seen in the axillary line when the light falls obliquely upon this area. During expiration a like process occurs but the relaxation of the lung produces less negative pressure;

therefore, the sign is not as well marked and often cannot be seen at all.

Litten's phenomenon can be seen in almost all subjects, but is best seen in the muscular adult male with a comparatively thin chest wall, and is least marked in the fat female. It is often well marked in elderly people but begins somewhat lower, due to the fact that the relaxed emphysematous lungs do not contract so completely in expiration.

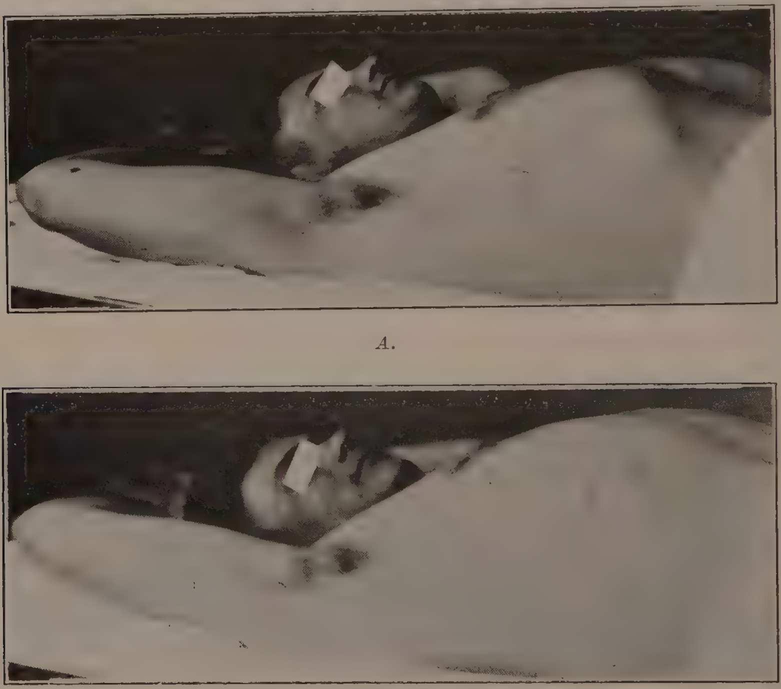


Fig. 69.—Litten's phenomena. *A*, showing position of the shadow at the beginning of inspiration; *B*, position of the shadow at the end of inspiration.

To elicit Litten's sign the patient is placed in the dorsal posture with the head comfortably elevated, and with the hands clasped above the head or the arms held slightly away from the thoracic wall. The feet are directed toward the source of illumination, the light from which should fall obliquely upon the side under observation. The examiner assumes a position near the feet of the patient, with his back toward the source of illumination. In a normal thorax the undulation is perceived to descend from about the seventh rib for the space of two inches or more during inspiration and, under favorable circumstances, to ascend to an equal degree during expiration.

Restriction of, or abolition of the "shadow" points to an abnormal condition within the thorax or abdomen which interferes with the free excursion of the diaphragm or to paralysis of this muscle. In the thorax it may signify pneumothorax, pneumonia, pleurisy with effusion, adhesions between the visceral and parietal pleura, hypertrophic emphysema, intrathoracic neoplasm or pulmonary tuberculosis, interfering with the proper inflation of the lungs. In the abdomen abolition of the undulation may be indicative of increased subphrenic pressure from hepatic or splenic enlargement, subphrenic abscess, ascites, or large abdominal tumor.

By marking the limits of Litten's sign and applying thin strips of lead over these marks with adhesive plaster, it will be found on fluoroscopic examination that the sign gives accurate information as to the diaphragmatic movements. Students will find this procedure an accurate check to their observations.

Pathologic Respiratory Variations.—In the presence of disease of the thoracic viscera and in many instances of disease of distant portions of the bodily economy, as well as in numerous constitutional affections, the respiration presents typical variations from the normal rhythm and type. The predominant change may be an increase or a diminution in the frequency or depth of respiration, or it may consist in a prolongation of one or the other phase of the respiratory cycle. In other instances the respiration becomes stridulous, sterterous, or it assumes the meningeal or Cheyne-Stokes type. In yet other instances the respiration becomes so labored as to be distinguished as dyspnea or orthopnea, which may or may not be attended by cyanosis.

Rapid Respiration (Polypnea).—Simple increase in the rate of the respiratory movements of the thorax is observed as a result of active physical effort, during mental and emotional excitement, and not infrequently is induced by the consciousness on the part of the patient of the examination, without possessing untoward significance. Febrile movement is attended by polypnea, as are certain forms of cerebral disease and hysteria. Pulmonary lesions which decrease the air space of the lungs as do the consolidations of pneumonia and tuberculosis; compression of the lung by pneumothorax, pleurisy with effusion, or mediastinal tumor; and elevation of the diaphragm by subphrenic abscess, ascites or abdominal neoplasm; all result in acceleration of respiration. During imperfectly compensated valvular heart disease the respirations are rapid; and during the course of diabetes mellitus and uremia elevation of the respiratory rate of varying degree is noted.

Slow Respiration (Oligopnea).—Diminution of the respiratory movements of the thorax, the number falling below fourteen respirations in the minute, is observed in states of collapse and in the coma of uremia or diabetes. Increased intracranial pres-

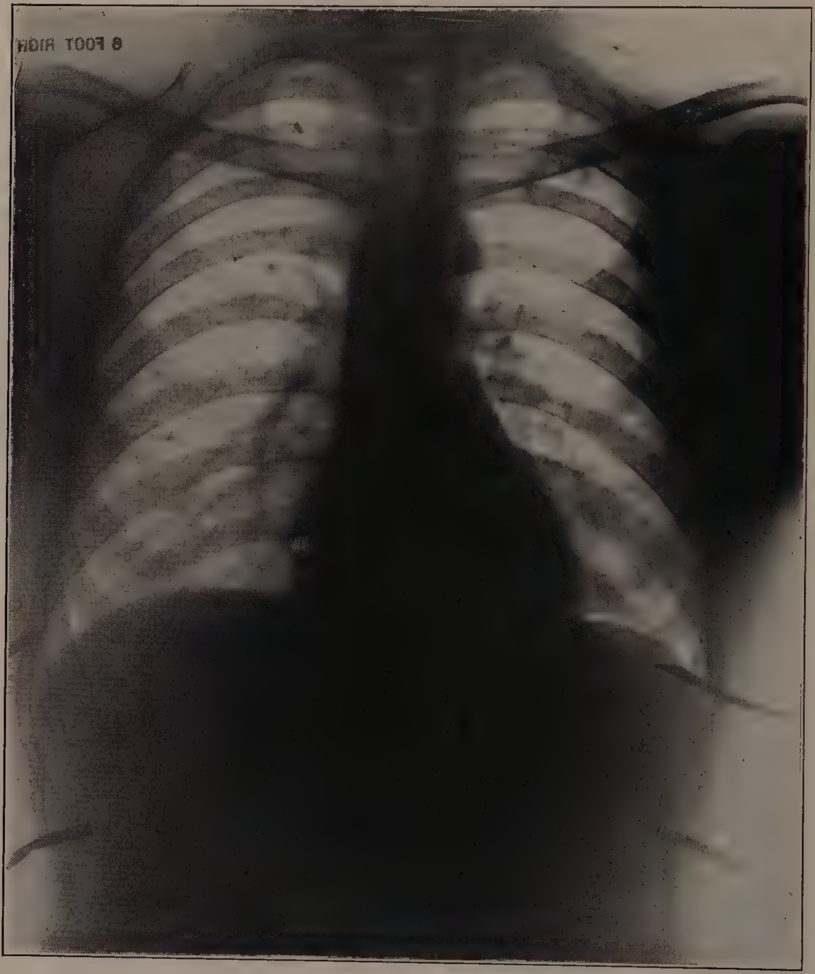


Fig. 70.—Lines on the radiograph are made by strips of lead pasted over lines marking out the limit of Litten's shadow.

Upper lead lines illustrate the beginning of the diaphragmatic shadow as noted on inspection. Lower lead lines illustrate the lower border of the diaphragmatic shadow as noted on inspection. Radiograph taken at the beginning of inspiration. Note how closely the diaphragmatic shadow approximates the upper lead line.

sure from tumor, abscess, hemorrhage, or meningitis is attended by an abnormally slow respiratory rate. The respiratory movements are also diminished during the course of infectious diseases which are associated with mental torpor. In these states, in addition to the diminution in the frequency of the respiratory movements, the rhythm of the respiration is usually disturbed.

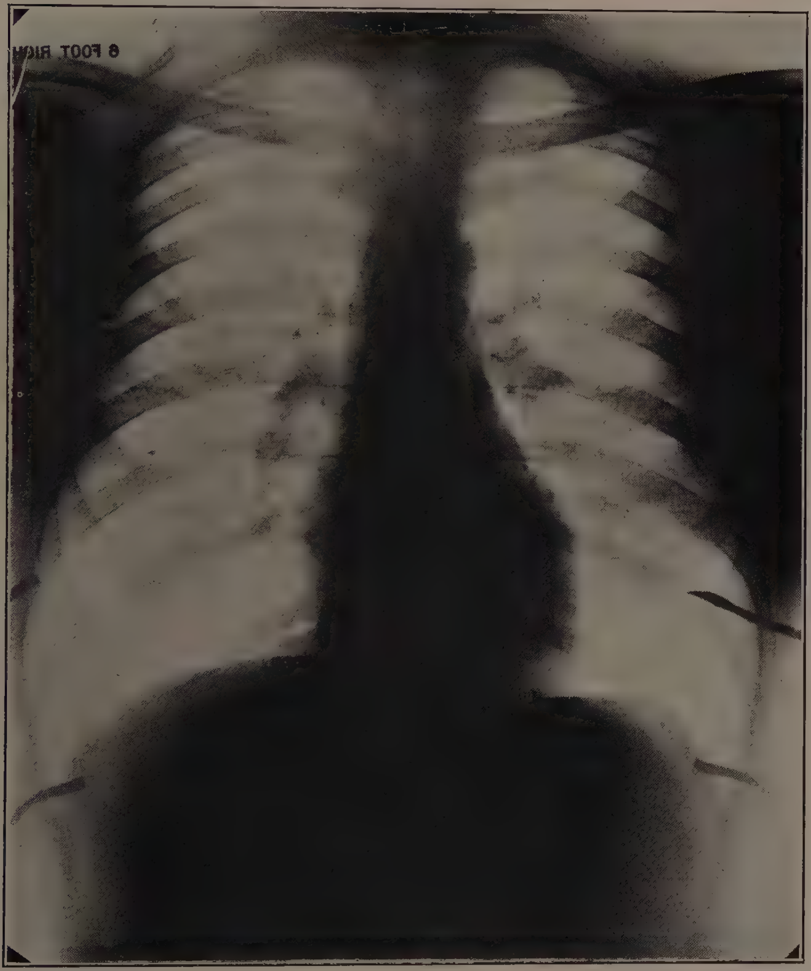


Fig. 71.—Upper lead lines illustrate the beginning of the diaphragmatic shadow as noted on inspection. Lower lead lines illustrate the lower border of the diaphragmatic shadow as noted on inspection. Radiograph taken at end of inspiration. Note how closely the diaphragmatic shadow approximates the lower lead line.

Prolonged Inspiration.—Inspiration is unduly prolonged in tracheal and laryngeal obstruction from spasm, compression, new growth, or foreign body. In these states the lower intercostal spaces and the upper abdomen are retracted during the violent inspiratory effort.

Prolonged Expiration.—The expiratory phase of the respiratory cycle is unduly prolonged in hypertrophic emphysema and in bronchial asthma, the muscles of expiration being called into play in the effort to expel the tidal air from the lung, substituting an active for a passive process. In the paroxysm of bronchial asthma, in addition to the prolongation, expiration is dotted with numerous râles.

Stertorous Respiration (Snoring Breathing).—During the coma of uremia, diabetes, and apoplexy respiration is frequently attended by stertor. The same type of respiration is frequently noted in cases of palatal paralysis. Less frequently stertorous respiration is encountered in the presence of large postpharyngeal abscess, with extensive adenoid vegetations in the nasopharynx, in chronic tonsillar hypertrophy, and in quinsy. Aside from these local causes, stertorous respiration is frequent in the coma resulting from poisoning from illuminating gas, alcohol, opium, or other narcotic drug.

Stridulous Respiration (Hissing Breathing).—Stridulous respiration is indicative of laryngeal or tracheal obstruction by infiltration, compression, new growth, or foreign body. It is produced by compression of these structures by enlarged glands, mediastinal tumor or an excessively hypertrophied heart. Hissing breathing also occurs in spasm and edema of the glottis, which is apt to develop in the course of syphilis or tuberculosis of the larynx, during diphtheria and acute laryngitis, or as a complication of one of the acute infectious fevers. Stridor also accompanies the paroxysms of pertussis and the attacks of laryngismus stridulus, the stridor in these instances being more pronounced during, or entirely limited to, inspiration. The stridor of laryngeal obstruction is as a rule accompanied by aphonia.

Cheyne-Stokes Respiration.—In this type of respiration, following a period of transient apnea, the respirations become progressively deeper until a maximum depth is attained, whereupon they gradually become more shallow to finally terminate in another apneic period. The period of apnea lasts from ten to twelve seconds as a rule, and during this time the patient is apt to become unconscious, with cyanosis, slowing of the pulse rate, and myosis. Persisting for a period of time varying from a few hours to several months, this type of respiration is of grave prognostic significance. It is frequently a sign of impending dissolution. Associated particularly with cerebrospinal meningitis, cerebral tumor, and apoplexy, Cheyne-Stokes respiration is also frequently noted during the coma of uremia and diabetes, and more rarely with arteriosclerosis and chronic valvular heart disease, and very rarely indeed it develops during the course of typhoid fever and lobar pneumonia.

Biot's Respiration (Meningeal Breathing).—In this type of pathologic respiration there are periodic interruptions in the respiratory sequence, during which the patient is apneic. The periods of apnea vary in duration and recur at irregular intervals. The periods of apnea lack the definite regularity which is noted in Cheyne-Stokes respiration. In addition, there is marked irregularity in the depth and rhythm of the individual respirations.

Occurring most frequently during the course of meningeal inflammation, Biot's respiration is also observed occasionally in cerebral hemorrhage and tumor, and during the periods of somnolence of acute infectious fevers.

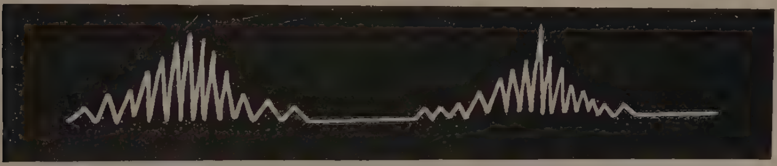


Fig. 72.—Cheyne-Stokes respiration.

Dyspnea.—Dyspnea, difficult or labored breathing, is recognized clinically by the increased frequency of the thoracic excursions and by the participation of the accessory muscles of respiration in the movements. Dyspnea may be present in varying grades, ranging from a slight increase in the frequency of the respirations to extreme difficulty, accompanied by blueness of the lips and finger-tips (cyanosis).

Dyspnea may be entirely limited to inspiration, as when the air passages are obstructed by a foreign body, or it may be expiratory as is the case in hypertrophic emphysema and bronchial asthma. Usually there is present a combination of inspiratory and expiratory dyspnea.

Dyspnea may arise as a result of deficient aeration of the blood content of the lungs arising as a result of an obstruction to the free ingress of air to the lungs, the causes of which are indeed varied. The obstruction may reside in hypertrophy of the turbinates or in adenoid vegetations in the nasopharynx, retropharyngeal abscess, or excessive tonsillar hypertrophy. The obstruction may reside in the larynx in the form of edema of the glottis, spasm of the rima glottidis, cicatricial stenosis from syphilitic laryngitis, or from lodgment of a foreign body in this portion of the air passages. Inspiratory dyspnea may also be caused by partial stenosis of the trachea, occurring as a sequence of cicatricial stenosis of the tube of intratracheal origin, or from compression of the trachea from without by an enlarged thyroid gland, a mediastinal tumor, or aneurysm of the aorta. Similarly, inspiratory dyspnea may arise from obstruction of a principal bronchus by cicatrix, pleural adhesions, or foreign bodies.

In all of these states the interference with respiration is inspiratory, as recognized by the prolongation of the inspiratory phase of the respiratory cycle. Inspiration is prolonged unduly, frequently with the development of an audible stridor, whereas expiration is brief and is attended by little movement of the thoracic walls.

Dyspnea involving principally expiration is noted in subjects of hypertrophic emphysema and during the paroxysm of bronchial asthma. In these diseases following upon a fairly rapid inspiration, there succeeds an unduly prolonged expiratory phase during which the inspired air is ejected tardily and with obvious effort upon the part of the subject of the disease. In bronchial asthma the expiratory phase is dotted with numerous piping sibilant and snoring, sonorous râles.

When the pulmonary alveoli are filled with the consolidations of pneumonia or phthisis, and when the lung is compressed by a large pleural effusion or mediastinal tumor, dyspnea develops as a result of an actual diminution of the aerating surface of the lung in which the circulating blood is exposed to the action of the aerial content of the lungs. A similar state obtains in the presence of extensive effusion into the peritoneum or in the presence of a large intraabdominal tumor or marked tympanites, conditions in which the diaphragm is displaced upward, partially immobilizing the thorax.

Dyspnea also develops in the presence of extreme weakness of the muscles concerned in the respiratory excursion of the thorax or with paresis of these muscles. A somewhat analogous state obtains when the thoracic excursions are voluntarily inhibited as a result of the pain incident to an acute fibrinous pleurisy or a fractured rib.

One of the most productive causes of dyspnea is to be found in valvular heart disease, in which regurgitant lesions of the left heart produce passive congestion of the pulmonary circulation. A similar state is induced by mechanical congestion of the lungs, in which the pressure of new growths or enlarged glands upon the pulmonary veins retards the flow of blood from the lungs to the heart. Under these circumstances dyspnea is extreme, amounting to orthopnea.

Acute infectious fevers associated with toxemia are productive of dyspnea, arising from the action of circulating toxins upon the respiratory centers. A more serious dyspneic state follows upon the action upon these centers of the toxins of uremia and diabetes, as evinced by the excessive dyspnea designated as the "air hunger" of Kussmaul.

Primary or secondary anemia, associated with a marked diminution in the hemoglobin or oxygen-carrying content of the blood occasions dyspnea, which may amount in certain cases to *orthopnea,* an extreme grade of dyspnea in which the subject is able to breathe only in the erect or sitting posture.

Abnormalities of Thoracic Expansion

Abnormalities in the degree of expansion of the thorax may be manifest in the form of bilateral or unilateral variations from the normal, or the anomaly may be circumscribed to certain, definite areas of the surface of the thorax. The normal degree of thoracic expansion is a variable quantity, varying with the degree of physical development of the individual and with the vital capacity of the lungs. The thorax of the athlete exhibits a power of expansion greatly in excess of that which is possessed by persons who habitually engage in sedentary occupations; and it is only by comparative study of various types of thorax that the examiner may form conclusive opinions as to what degree of thoracic expansion is to be considered abnormal in the individual case under investigation.

Bilateral Variations.—Increased general expansion of the thorax in all of its diameters is observed after active physical effort and during violent emotional excitement without possessing untoward significance. In hypertrophic emphysema, and in the course of a paroxysm of bronchial asthma, as well as in purely costal respiration due to abdominal disease, a general increase in the expansion of the thorax is the rule.

Decreased general expansion of the thorax in its several diameters is characteristic of the small chest of elderly patients with atrophic emphysema. A similar decrease in expansion occurs as a result of general muscular weakness and in paralysis of the intercostal muscles. Obstruction of the upper air passages by limiting the free ingress of air to the lungs is attended by limitation of the thoracic expansion; and bilateral pleurisy with effusion and the bilateral consolidation of double pneumonia result in a similar diminution of the inspiratory excursion of the chest wall. The pain of diaphragmatic pleurisy, pleurodynia, and intercostal neuralgia causes a general diminution of thoracic expansion. Scleroderma affecting the chest wall and premature calcification of the costal cartilages result in general limitation of expansion.

Unilateral Variations.—Increased expansion of one side of the thorax occurs in the presence of vicarious expansion of one lung in compensation for a crippling of its fellow. The cause of the imperfect expansion of the unsound lung may reside in the lung itself in the form of tuberculous or pneumonic consolidation, the collapse of atelectasis, or the fibrosis of chronic interstitial pneumonia, fibroid phthisis, or pulmonary syphilis; or, on the contrary, the cause of malfunction may be extraneous to the lung, and may assume the form of pulmonary compression by pleural effusion, pneumothorax, or mediastinal tumor.

Decreased expansion of one side of the thorax is noted as a consequence of diminution of the air space of the lung by the consolidation of phthisis or pneumonia or the fibrosis of chronic interstitial pneumonia, pulmonary syphilis, or fibroid phthisis. Stenosis of a principal bronchus, resulting in extensive atelectasis, leads to diminution or abolition of expansion upon the corresponding side of the thorax. Pleural effusion, pneumothorax, or tumor of the lung or pleura, impairs the thoracic expansion upon the side of the disease and results in compensatory expansion of the opposite side. Unilateral diaphragmatic paralysis, as well as increased subphrenic pressure due to extensive enlargement of the liver or spleen and the traction of pleural adhesions, cause limitation of thoracic expansion upon one side.

Unilateral variations of expansion are more readily detected upon inspection of the thorax than are bilateral changes. In cases in which only a slight departure from normal expansion is in evidence, the deficiency is most readily detected by drawing a line upon either side of the thorax below the clavicle and inspecting the anterior thoracic surface from a position behind the patient.

Not infrequently the lesion which is responsible for the deficient expansion produces deformity of the thorax as well, which in certain instances is characteristic of the cause. In large pleural effusion and in pneumothorax, in addition to the unilateral immobilization of the thorax, there is unilateral bulging of the chest wall. In limitation of expansion due to pleural adhesions and to advanced phthisis, on the contrary, there is apt to be unilateral retraction of the thorax. Moreover, a unilateral retraction which is most marked in, or which is limited to, the upper thorax is suggestive of phthisis, whereas a similar retraction of the lower anterolateral thorax points rather strongly to postpleuritic adhesions as the cause.

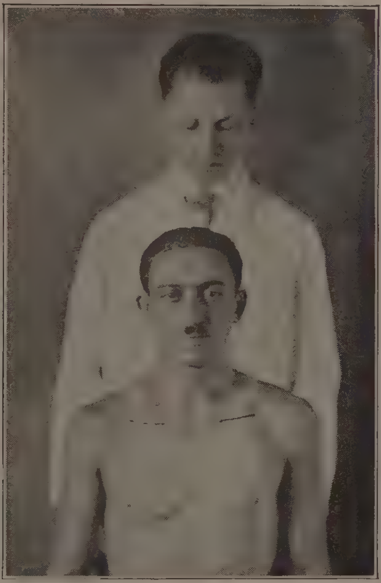


Fig. 73.—Detection of lagging of apices.

Local Variations.—Circumscribed areas of increased thoracic expansion are encountered over the apices and the bases of the lungs, and the location of the vicarious expansion in these cases is of potent diagnostic import. Increased expansion over a pulmonary apex, limited to the supraclavicular and infraclavicular regions, occurs in the consolidation of lobar pneumonia involving the lower lobe of the lung, and in compression of the lung by pleurisy with effusion, mediastinal tumor, or excessively hypertrophied heart. In the presence of extensive tuberculous consolidation of the apex, on the contrary, the lower portion of the thorax expands vicariously in compensation for the deficient expansion of the upper lobe of the lung.

Local or circumscribed diminution in the degree of expansion of the thorax at the pulmonary apex in the supraclavicular and infraclavicular regions is noted in apical pulmonary tuberculosis. A similar limitation in expansion of the lower anterolateral regions of the thorax is commonly due to the traction of pleural or of pleuropericardial adhesions.

Wavy expansion of the thorax is occasionally encountered in connection with lobar pneumonia, when successive areas of the thorax appear to expand in an irregular sequence, one area expanding prior to the expansion of adjacent areas.

Inspiratory retraction of the lower intercostal spaces in the lower axillary and infraaxillary regions is a normal phenomenon in the first half of the inspiratory phase of the respiratory cycle. During the first half of the act of inspiration the interspaces are moderately retracted in this area, to become flattened out upon the same plane as the ribs during the second half of the inspiratory act, but never exceeding this level throughout the respiratory cycle. Bäumler holds that this initial respiratory retraction of the interspaces in the normal subject is due to the contraction and descent of the diaphragm prior to the contraction of the intercostal muscles, in this wise creating a transitory diminution in intrathoracic pressure or a negative thoracic pressure, with the result that the atmospheric pressure exerted upon the exterior of the thorax during the initial stage of the inspiratory act causes recession of the lower interspaces at this time.

Pathologic inspiratory recession of the lower intercostal spaces differs from this normal inspiratory recession in the fact that the period of the recession consumes the entire time of the act of inspiration. The distribution of the retraction possesses definite localizing value in these cases. As the obvious cause of the retraction in this case is the inability of the lung to become fully inflated with air, if the retraction is bilateral, affecting both lungs to an equal degree, the seat of obstruction is situated above the tracheal bifurcation; whereas, if one entire side of the thorax exhibits pathologic inspiratory retraction of the intercostal spaces, the seat of the stenosis is evidently in one of the primary bronchi. Circumscribed areas of inspiratory retraction signify stenotic lesions of the smaller bronchial tubes.

MOVEMENTS OF THE THORAX DUE TO CARDIOVASCULAR ACTIVITY

The Cardiac Impulse

After inspection of the various phenomena connected with the respiratory system, the examiner considers the visible evidences of activity of the cardiovascular mechanism. The so-called apex impulse is in reality the impact of the lower portion of the right ventricle against the chest wall with each ventricular systole. The true apex of the heart is about one-half inch downward and to the left of the maximum impulse. In the normal subject the point of maximum intensity of the impulse is seen in the fifth intercostal space, about three and one-half inches from the midsternal line. In the sthenic type it is usually a little more to the left and in the asthenic type a little more to the right and somewhat lower, often under the sixth rib. In children it may be noted as high as the fourth intercostal space.

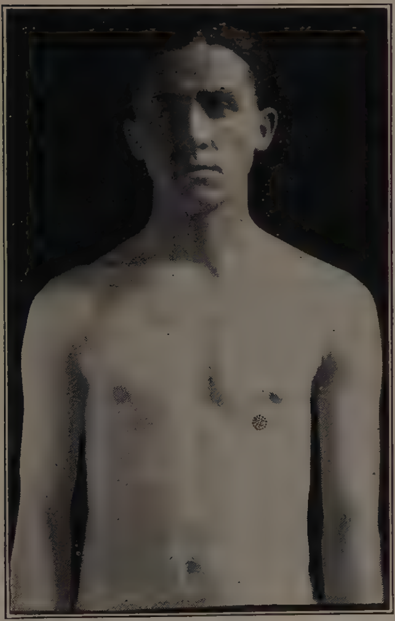


Fig. 74-*A*.—Site of normal cardiac impulse.

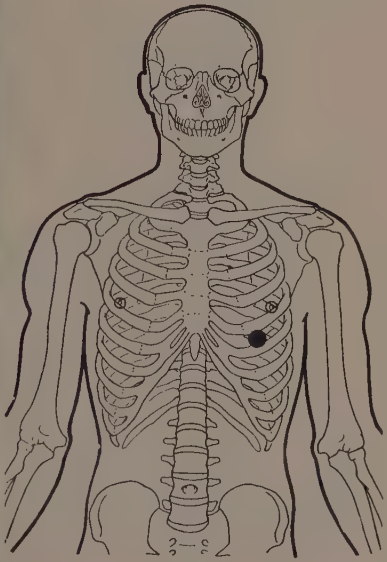


Fig. 74-*B*.—Site of normal cardiac impulse.

When lying on the left side the impulse moves about three-fourths of an inch to the left and when lying on the right side about one-half inch to the right. The normal cardiac impulse covers an area corresponding to the interspace in which it lies, with a lateral diameter of about one inch. In the female the mammary gland may totally obscure the visible impulse. The impulse may also be obscured in subjects with excessively fat chest walls or with very voluminous anterior pulmonary borders. The impulse is best seen with the subject in the erect posture bending slightly to the left. Cross marks with a soft pencil over the apex region may help the observer to detect a faint impulse.

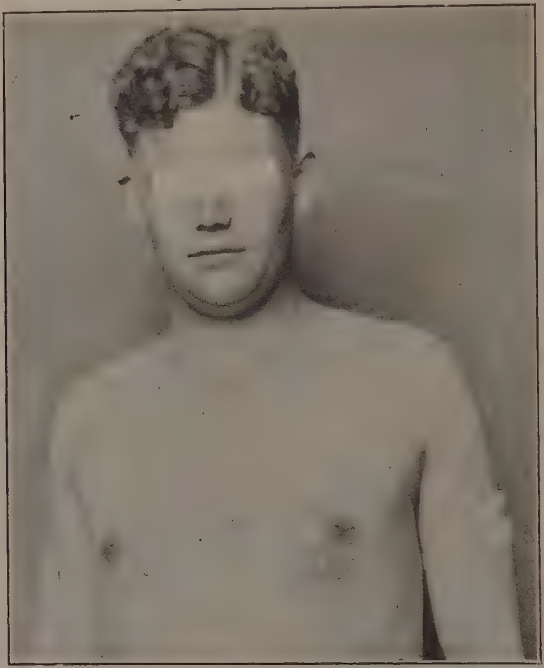


Fig. 75.—Cross lines drawn with soft pencil over the region in which the cardiac impulse usually appears will often enable the observer to detect it more easily.

A small scrap of paper attached to the apex region by moistening will aid in visualizing the pulsation and will accentuate any variations in its time and force.

The impulse becomes more pronounced after violent exercise or emotional excitement. It is usually better seen at the end of expiration when the lung is retracted, allowing a greater portion of the heart to come in contact with the chest wall.

In disease the cardiac impulse may be more marked in cardiac hypertrophy due to valvular disease, especially aortic and mitral regurgitation, increased peripheral resistance, as in arterial hypertension, or acute febrile diseases. The impulse becomes weaker following fatigue of heart muscle from acute strain or degeneration of the muscle from chronic overwork, infection, or sclerosis.

The weak impulse is evidenced by its shorter and more slapping character. This sudden tap against the chest wall is often more easily visualized than the slow deliberate push of the more vigorous ventricular contraction.

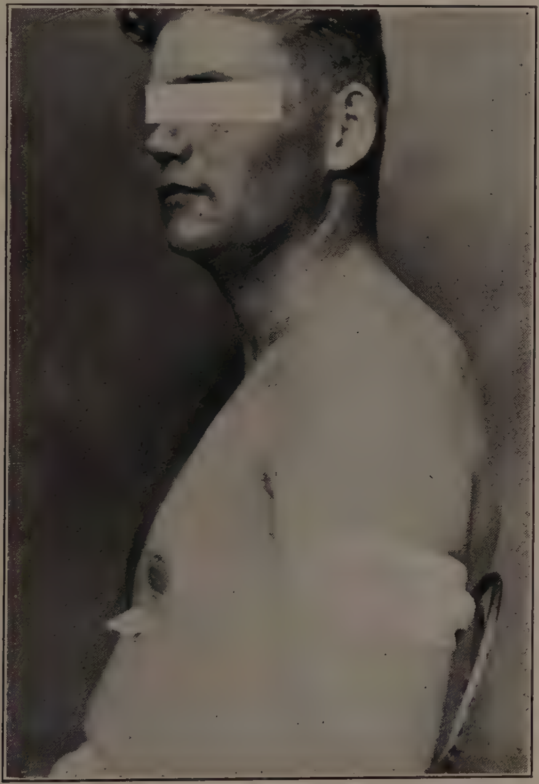


Fig. 76.—A scrap of paper bent at right angles and applied to the cardiac impulse. The lever thus produced will exaggerate the amplitude of the impulse and accentuate any changes in its time or force.

The impulse may be obscured in emphysema which interposes a thick cushion of lung between the heart and the chest wall, by fluid or air in the left pleural cavity, or by fluid or air in the pericardial sac.

Displacement of the Cardiac Impulse

Aside from the physiological displacement to which the cardiac impulse is subject, various abnormal displacements of the impulse may be encountered, arising from endocardial and exocardial causes. Under these circumstances the direction and degree of apical displacement not infrequently furnish a clue to the cause of the displacement.

Upward Displacement of the cardiac impulse is noted in the presence of cardiac atrophy, in the presence of diaphragmatic hernia, and when the diaphragm is elevated as a result of increased intraabdominal tension. In the presence of fibroid retraction of the anterior border of the left lung the impulse is elevated, and it is commonly visible over an extensive area along the left sternal border. The pressure of an extensive effusion into the right pleural sac occasionally causes the liver to rotate about its axis in such a manner as to tilt the narrow left lobe of the liver upward, resulting in elevation of the superjacent heart and displacement of the cardiac impulse upward and toward the left.

Downward Displacement of the cardiac impulse occurs as a consequence of the progressive development of an aneurysm of the aortic arch or of a mediastinal tumor which presses upon the base of the heart. A similar displacement frequently attends hypertrophic emphysema, partially as a result of pressure exerted upon the heart by the voluminous anterior borders of the lungs in this disease, and partially as a result of the inspiratory position of the thorax peculiar to this disease, in which the ribs tend to move upward over the heart, simulating a downward displacement of the heart.

Extensive pericardial effusion, prior to the obliteration of the cardiac impulse, is apt to be attended by depression of the diaphragm, in which event the cardiac impulse is displaced downward.

In addition to these exocardial pulsion and traction displacements, the cardiac impulse is displaced downward and toward the left in left ventricular hypertrophy and dilatation, the impulse under these circumstances not infrequently occupying the seventh or eighth interspace external to the left midclavicular line.

Displacement to the Left.—In addition to the left lateral displacement of the cardiac impulse which attends left ventricular hypertrophy and dilatation, a further important cause of such displacement is right ventricular hypertrophy and dilatation, in which the apex beat is frequently localized external to the left midclavicular line. Horizontal displacement of the impulse toward the left likewise occurs as a consequence of the pressure exerted by the progressive accumulation of fluid or gas in the right pleural cavity, and as a result of the traction of adhesions between the pericardium and left pleura.

Fibroid retraction of the anterior border of the left lung results in left lateral displacement and elevation of the cardiac impulse; and a similar displacement of the impulse attends hepatic enlargement and gastric distention. In the presence of pericardial effusion of moderate degree the apex beat is frequently elevated and displaced toward the left.

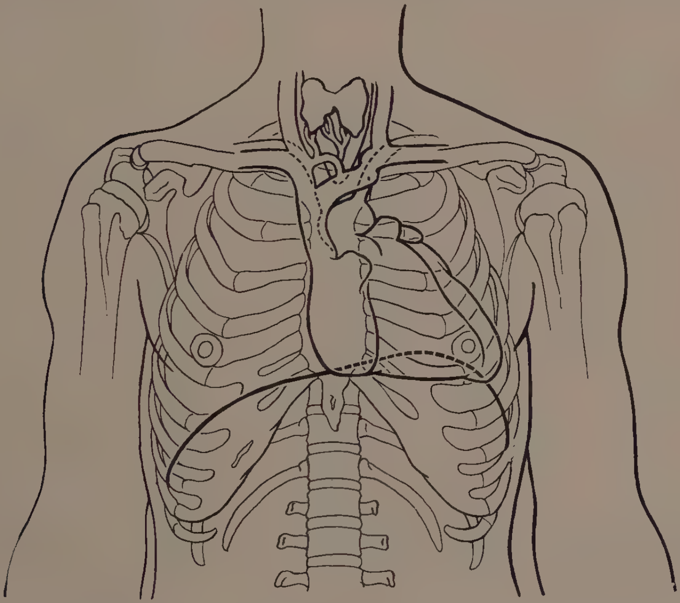


Fig. 77.—Illustrating moderate displacement of the heart toward the left in compensatory emphysema of the right lung, with elevation of the left vault of the diaphragm and displacement toward the left of the structures in the median line of the neck.

Massive collapse of the left lung will displace the cardiac impulse to the left.

Displacement to the Right.—The cardiac impulse is displaced toward the right by the pressure of fluid or gas in the left pleural cavity, and it is drawn to that side by the traction of right pleuropericardial adhesions. Compensatory emphysema of the left lung, when sufficient in degree, may displace the impulse toward the right. Similarly, the progressive development of a neoplasm of the left lung is capable of causing apical displacement toward the right side of the thorax. In the presence of congenital transposition of the thoracic viscera the cardiac impulse is encountered in the fifth right intercostal space.

Massive collapse of the right lung will displace the cardiac impulse to the right.

Apical Retraction.—When the pericardium is adherent to the parietal chest wall, one may see instead of an impulse a systolic retraction. Congenital or rachitic deformities of the chest wall may cause a similar retraction. Occasionally in cases of hypertrophy of the left ventricle the cardiac systole diminishes the cardiac volume sufficiently to actually increase the negative pressure in the left pleural cavity, thus producing a systolic retraction from atmospheric pressure.

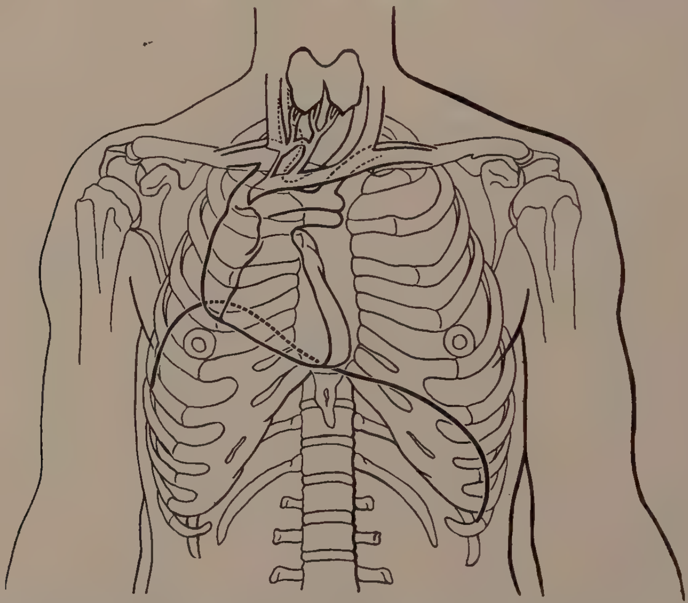


Fig. 78.—Illustrating cardiac displacement toward the right in compensatory emphysema of the left lung, which is attended by depression of the diaphragm upon the left side and by displacement of the structures of the median line of the neck toward the right side.

Extraapical Pulsation

Abnormal areas of pulsation along the borders of the precordia or in more remote regions are noted during the course of cardiovascular disease; and they possess a variable significance, depending upon their sites and their relations to the events of the cardiac cycle.

Pulsation at the Base.—Visible pulsation at the base of the heart, which is synchronous with ventricular systole, is indicative in the vast majority of cases of aneurysm of the transverse portion of the aortic arch. As a rule, when visible pulsation is present in this region the aneurysm has eroded the sternum, and is attended by pain of a dull, boring character. With less constancy pulsation at the base is referable to extreme cardiac hypertrophy or dilatation.

Pulsation at the Right Sternal Border.—Systolic pulsation in the first and second intercostal spaces adjacent to the right sternal border accompanies aneurysm of the ascending aorta. Systolic pulsation in the second, third and fourth interspaces occurs when the heart is displaced toward the right by the pressure of fluid or gas in the left pleural sac, or by the traction of adhesions between the pericardium and the right pleura.

Presystolic pulsation in the same area occurs in the presence of right auricular dilatation, and occasionally when the heart is exposed to the thoracic wall as a result of fibroid retraction of the anterior border of the right lung.

Pulsation at the Left Sternal Border.—Systolic pulsation adjacent to the left sternal border in the second and third interspaces, most pronounced between the left sternal and parasternal lines, accompanies aneurysm of the descending portion of the aortic arch. Fibroid retraction of the anterior border of the left lung or displacement of the heart toward the left causes the systolic pulsation of the conus arteriosus to become visible in the second, third and fourth interspaces adjacent to the sternum.

Presystolic pulsation in the second left interspace adjacent to the sternum points to dilatation of the left auricle as the causative lesion.

Episternal Pulsation.—The student of physical diagnosis is early impressed by the great frequency with which pulsations are encountered in the episternal notch. Frequently it is not easy to assign the pulsation to its proper cause, as pulsation in this restricted space is encountered in a variety of conditions.

Episternal pulsation is a normal phenomenon in many elderly subjects; it occurs as a sign of essential or secondary anemia; it follows excessive physical effort or the ingestion of stimulants; and it is prone to develop during the course of cardiac neuroses. The pulsation may be caused by the presence of a cervical rib, or it may be due to an abnormal course of the great vessels at the root of the neck. A similar systolic pulsation is encountered in this region when the subclavian artery is exposed to the thoracic wall by fibroid retraction of the pulmonary border.

But it should be borne in mind that systolic pulsation of the episternal notch attends simple dilatation and aneurysm of the transverse portion of the aortic arch with great constancy.

Supraclavicular Pulsation.—Pulsation in the supraclavicular region may be of *arterial* or *venous* origin. Systolic pulsation in the right supraclavicular region is noted in connection with aneurysm of the innominate or subclavian artery, and the pulsation is usually accompanied by a palpable thrill. In the presence of left ventricular hypertrophy, with or without regurgitation at the aortic orifice, there is frequently bilateral *carotid pulsation* in the supraclavicular region. In this instance it can be demonstrated that the systolic pulsation occurs over the course of the carotid artery, along a line drawn from the sternoclavicular articulation to a point equidistant between the angle of the jaw and the mastoid process. Carotid pulsation is likewise encountered in the presence of aneurysm of the aortic arch, arteriosclerosis, exophthalmic goiter, anemia, and after the ingestion of stimulants.

A pulsation is often seen just below the outer third of the clavicle on both sides of the chest. This is due to the pulsation of the axillary artery and not, as is frequently supposed, to the subclavian artery. It is best seen in subjects with thin chest walls, and is increased after exercise. It may be seen in both the upright and recumbent positions.

Thoracic Retraction (Broadbent's Sign)

In chronic adhesive pericarditis, in the presence of extensive adhesions between the pericardium and the diaphragm, with each ventricular systole there is noted a systolic retraction of the thoracic wall. Broadbent directed attention to the frequency with which systolic retraction is visible upon the left side of the thorax posteriorly, below the angle of the scapula in the tenth and eleventh interspaces in this disease. There is frequently to be noted a similar systolic retraction of the anterior chest wall in the eighth and ninth interspaces in the left parasternal line.

CHAPTER XIII

INSPECTION OF THE ABDOMEN

Technic.—During inspection of the abdomen the patient should in the first instance assume the dorsal decubitus, the position in bed or upon the examining table being absolutely symmetrical and free from undue tension of the abdominal muscles. The room temperature should be warm enough to prevent muscular contraction and shivering. The subject should be covered with a sheet which may be turned down, exposing the abdomen freely to the pubic symphysis. The examiner should in the first place stand beside the abdomen and inspect its surface first by direct illumination and afterward by oblique illumination, the latter frequently revealing slight pulsations, protrusions or vermicular movements which escaped detection during the examination by direct illumination. The examiner should then assume, consecutively, positions near the feet and near the head of the subject and should inspect the abdominal wall from these two positions.

For the purpose of detecting certain phenomena such as the vernicular movements of visible peristalsis and abdominal sagging due to visceroptosis, the abdomen should in addition be inspected with the subject in the standing posture. Finally, in certain instances it is desirable to examine the patient in the knee-chest position, a position which permits movable tumors of the abdominal viscera to fall forward and to become more clearly visible upon inspection of the abdominal surface.

THE SKIN OF THE ABDOMEN

The integument of the abdomen is lax and loose in the presence of emaciation from chronic wasting disease, and is tense and glistening in the presence of abdominal distention from ascites, pregnancy, or increased intraabdominal tension attending the development of a large intraabdominal tumor. Whitish or silvery striæ, *linœ albicantes,* distributed over the lower portion of the abdomen and the upper portions of the thighs are indicative of former abdominal distention, whether from tumor, ascites, or pregnancy.

Scars upon the abdomen may result from former surgical operations, from trauma, or from the eruption of syphilis or other cutaneous disease. A scar in the groin is suggestive of suppuration of an inguinal gland, which has opened spontaneously or has been opened by the surgeon.

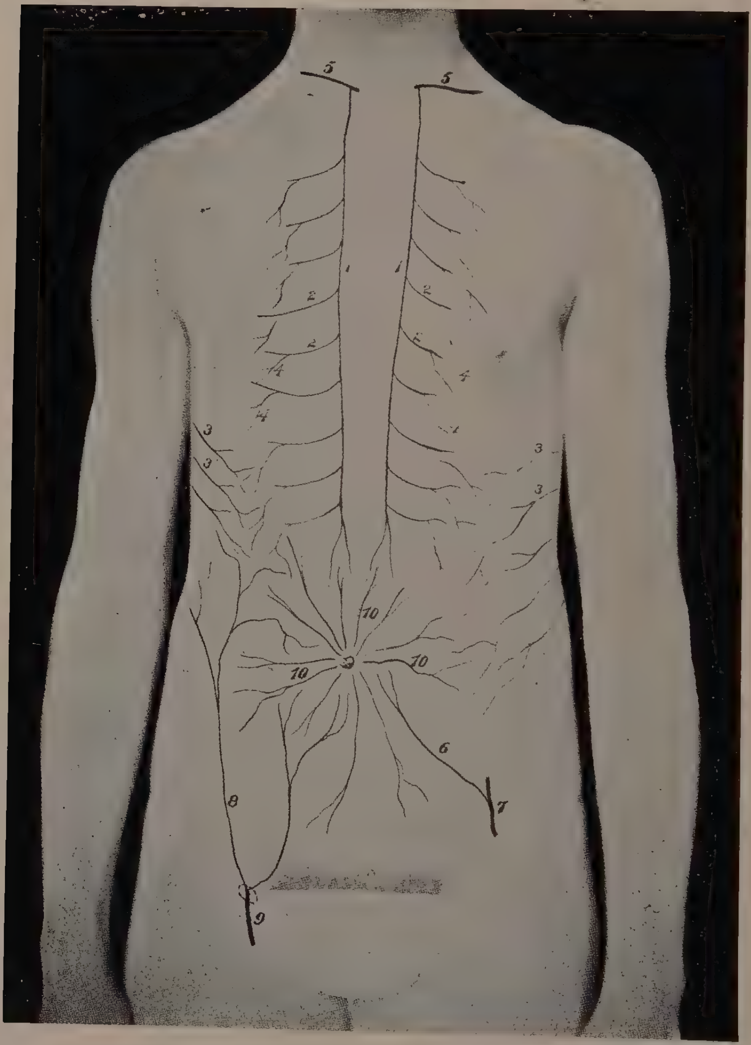


Fig. 79.—Establishment of collateral circulation in portal vein obstruction and mediastinal tumor. (Eisendrath.)

1, internal mammary veins; *2*, anterior intercostal veins; *3*, posterior intercostal veins; *4*, radicles of *2*; *5*, subclavian vein; *6*, deep epigastric vein; *7*, external iliac vein; *8*, superficial circumflex iliac vein; *9*, internal saphenous vein; *10*, caput medusae.

The *linea nigra*, a line of brown pigment in the median line of the abdomen below the umbilicus, accompanies pregnancy and chronic abdominal distention from other cause. It is usually more noticeable in brunettes.

The abdomen is the usual site of the cutaneous eruption of typhoid fever, the *rose spot* of this disease. These spots are small, discrete, hyperemic, slightly elevated papules, which readily disappear upon pressure. In certain instances the papule is surmounted by a small vesicle. They are not limited to the abdomen, though most frequently encountered in this locality; but may appear upon the back, the arms, or the thighs. The rose spots appear in successive crops, disappearing in two or three days, sometimes leaving a brownish stain.

During pregnancy the abdominal skin occasionally exhibits the brownish areas of chloasma.

THE SUPERFICIAL VEINS OF THE ABDOMEN

In most normal subjects the superficial abdominal veins can be seen. Their course is not constant and the number varies considerably.

In the presence of portal obstruction, there is present upon the abdominal wall a series of distended superficial veins extending outward from the umbilicus in a radial manner, constituting the *caput medusæ*. A distention of the superficial veins over the abdomen, communicating with similarly distended veins over the thorax, is indicative of portal vein obstruction by hepatic cirrhosis or tumor, chronic ascites, or pressure exercised upon the inferior or superior vena cava by abdominal or mediastinal tumor. The direction of the blood current in the distended vein is an index to the site of the obstruction. If, upon compression of the vein, it is found that the direction of the blood current is upward, the obstruction is in the portal vein or inferior vena cava; whereas, if the current is directed downward, the obstruction is situated in the course of the superior vena cava. The venous tortuosity under these circumstances is an evidence of the effort at the establishment of collateral circulation in the presence of obstruction of the usual venous channels.

THE UMBILICUS

The umbilicus should be examined for protrusion, retraction, skin eruptions, and inflammation. A protruding umbilicus is noted in umbilical hernia, during the latter months of pregnancy, in the presence of portal vein obstruction, ascites, or abdominal distention due to the development of a large intraabdominal tumor. In the obese subject on the contrary the umbilicus is markedly retracted.

PULSATIONS

Hepatic Pulsation.—Systolic pulsation of the liver accompanies tricuspid regurgitation and is apt to be attended by signs of general venous stasis, as edema of the extremities, ascites, and cyanosis. Not infrequently a hepatic pulsation which is not visible is yet distinctly palpable upon bimanual palpation of the hepatic region. True expansile pulsation of the liver, which occurs with incompetence of the tricuspid valve, must be differentiated from the impulse which is frequently transmitted to the liver by the impact of an overacting right ventricle.

Epigastric Pulsation.—Systolic pulsation of the epigastrium is occasionally noted in the normal subject without possessing untoward significance. Under these circumstances it is usually to be attributed to physiologic bathycardia or to shortness of the sternum. Systolic epigastric pulsation likewise accompanies hypertrophy of the right ventricle, and is also noted when the heart is displaced toward the right so that the apex lies behind the sternum.

Diastolic pulsation of the epigastrium accompanies anemia and neurasthenia, and is noted in subjects of chronic gastric indigestion with great frequency. Diastolic pulsation in this region likewise accompanies a tumor of an abdominal organ overlying the aorta, the pulsation being transmitted to the tumor during each pulsation of the artery.

Epigastric pulsation arising from the abdominal aorta occupies a position in the lower epigastrium and is situated slightly to the left of the median line, whereas the epigastric pulsation of right ventricular hypertrophy occupies the superior portion of the epigastrium in the costal angle.

VISIBLE PERISTALSIS

The peristaltic movements of the stomach, small intestine, or large intestine at times become visible as a vermicular movement upon the anterior abdominal wall. In its exaggerated form it is indicative of obstruction located at the pylorus, at some point in the course of the small intestine, or in the colon.

The peristaltic movement of the stomach is visible under these circumstances in the upper portion of the abdomen, pursuing a direction from left to right and somewhat downward in the epigastric region.

Visible peristalsis of the small intestine is chiefly confined to the umbilical region, whereas that occasioned by obstruction of the large intestine is observed over the course of the colon. When it is not unduly pronounced, peristaltic movement may often be accentuated by applying a cold hand to the abdominal surface or by flicking the surface of the abdomen with a towel which has been wet in cold water. When the site of the obstruction is situated in the ileum just proximal to the ileocecal valve, the visible peristalsis assumes frequently a "ladder pattern," the waves lying one above the other in the umbilical region.

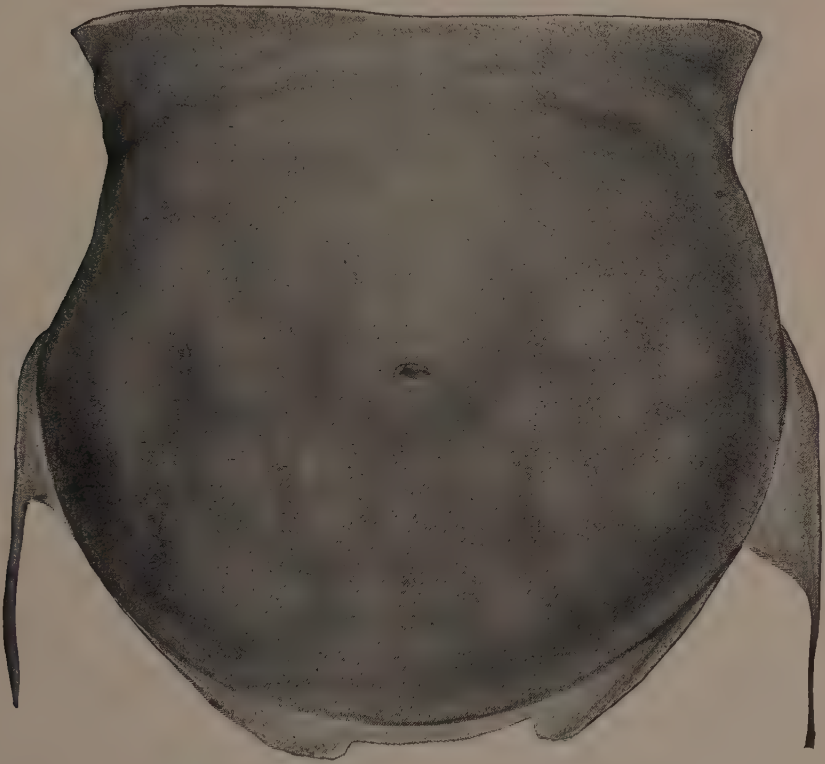


Fig. 80.—Normal intestinal peristalsis. (Austin, after Nothnagel.)

Visible peristalsis observed in extremely emaciated patients with extremely thin abdominal walls, or in women in whom repeated pregnancy has caused a diastasis of the rectus muscles, possesses no diagnostic significance.

RESPIRATORY MOVEMENTS OF THE ABDOMEN

In the normal subject the abdomen becomes more prominent during inspiration, due to descent of the diaphragm increasing the intraabdominal pressure. This phenomenon begins in the epigastric angle and extends over the entire abdominal surface. During expiration a consequent recession begins below the umbilicus and ends in the epigastric angle. It is more prominent in the diaphragmatic type of respiration.

Fixation of the abdominal wall with inhibition or abolition of the respiratory movements is indicative of pain arising within the abdominal cavity with the respiratory excursion of its walls. which is not infrequently due to acute peritonitis, paralysis of the diaphragm will also abolish respiratory movements of the abdomen.

CONTOUR OF THE ABDOMEN

The normal abdomen is symmetrical with a moderate degree of anteroposterior flattening in the male subject. The walls are of uniform tension. The umbilicus of the normal abdomen is neither unduly depressed nor protruding. The normal abdomen falls away from the costal margin, becomes gradually more prominent to a point about one and one-half inches below the umbilicus, and then becomes less prominent as it approaches the pubis.

Variations in the contour of the abdomen may be symmetrical or asymmetrical. Symmetrical enlargement of the abdomen is encountered in the presence of obesity, pregnancy, intestinal meteorism, ascites, and in the presence of visceroptosis. Symmetrical retraction of the abdomen is encountered in conditions of emaciation depending upon cardiac or pyloric stenosis, or the moribund state.

Obesity

In the obese subject the abdomen assumes a contour which is characteristic of the condition. The enlargement is symmetrical; the normal cutaneous flexion folds are accentuated; the umbilicus is definitely retracted; and the inferior regions of the abdomen are pendulous, encroaching to a variable extent upon the thighs and the pubes. The thick abdominal wall in this state may be identified by pinching it up between the thumb and forefinger, or by approximating the two palms after depressing either side of the abdomen.

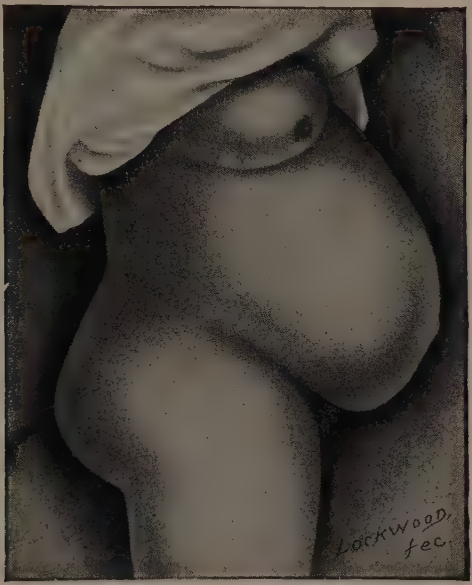


Fig. 81.—Obesity, mistaken for pregnancy by patient. (Crossen, after Williams.)

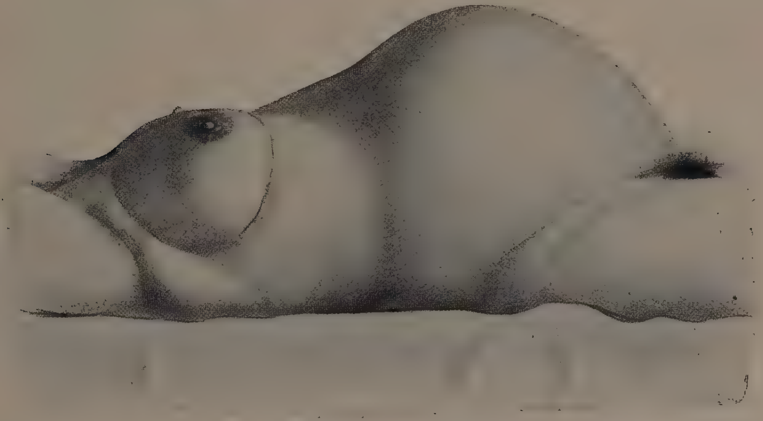


Fig. 82.—Contour of the abdomen in pregnancy with patient recumbent. (Crossen, after Edgar.)

Pregnancy

The abdominal enlargement accompanying pregnancy is progressive, increasing gradually as the uterus rises out of the pelvis, and comes to occupy the abdominal cavity. In its fully developed state the umbilicus protrudes, the abdominal skin presents the lineæ albicantes of pregnancy, and the abdominal distention is accompanied by changes in the breasts and the positive signs of pregnancy. The degree of abdominal distention is much more considerable in the case of multiparous women than is the case with primiparæ.

Meteorism

Meteorism, or tympanites, produces symmetrical abdominal enlargement which is of transient duration. During the attack the abdominal walls are tense, smooth and shiny. Not infrequently the peristalsis of the stomach or intestine is visible upon the tense abdominal wall. The findings in such cases are not altered by changes in posture. The umbilicus is level with the adjacent integument, or it actually protrudes.

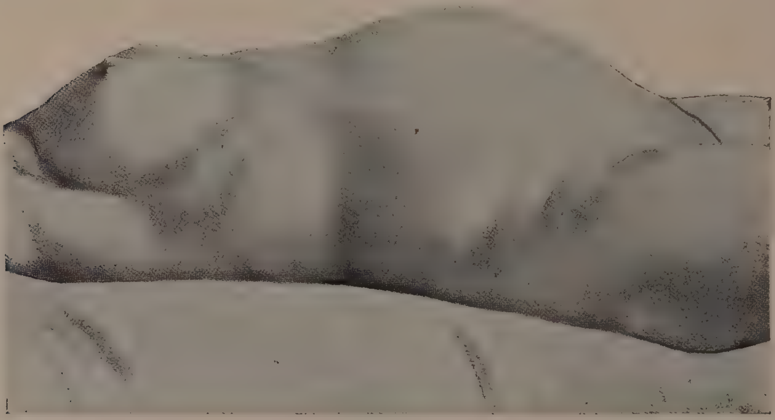


Fig. 83.—Tympanites, mistaken for pregnancy by the patient. (Crossen, after Edgar.)

Ascites

The degree of abdominal enlargement accompanying ascites varies with the amount of fluid in the peritoneal cavity. With the development of the ascitic fluid there is a gradual and uniform enlargement of the abdomen. The contour of the abdomen is characteristically altered, the anteroposterior diameter increasing with moderate flattening of the lateral regions. In the presence of an extensive effusion into the peritoneal cavity the abdominal skin is smooth and tense, and not infrequently it presents silvery striae. Enlarged, tortuous superficial veins are frequently in evidence. The umbilicus is level or protruding.

Gastroptosis

In gastroptosis, with the patient in the erect posture, the abdomen presents a bulging which is most pronounced in the umbilical region, and which is accompanied by more or less flattening of the epigastric region and the flanks. In the presence of gastrectasis, however, the flattening of the epigastrium is not striking. The groins commonly present a furrow, above which is to be noted a local bulging of the abdominal wall which has been likened to a sausage.

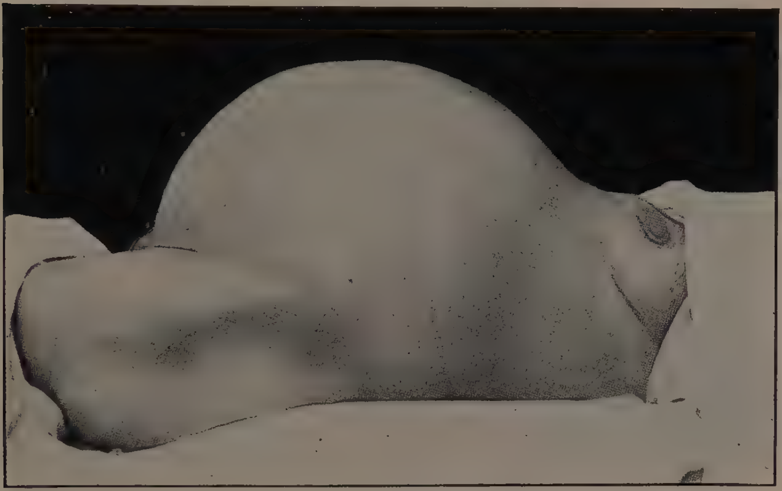


Fig. 84.—Extreme ascites. In the patient from which this photograph was taken, the abdomen was so distended with fluid that the wall was raised higher than the mesentery would permit the intestine to float, giving dullness about the umbilicus as well as elsewhere. The rise of the wall from below is rather abrupt. There is also edema of the wall, as shown by the persisting groove where the skirts were tied about the waist. (From Crossen.)

With the patient in the recumbent posture the epigastrium is depressed to a variable degree, and visible or palpable pulsation is communicated to the abdominal wall from the subjacent aorta.

Enteroptosis

In enteroptosis, which is commonly attended by similar falling of the stomach, liver and right kidney (Glenard's disease), with the patient in the upright posture the abdominal contour as viewed from the front is irregularly pear-shaped, resembling a sagging sack or a gourd. The ventral wall of the abdomen slopes gently downward to the central umbilical region, in which it bulges in all directions, approaching or overlapping the pubes inferiorly. In the groins the abdomen exhibits a fairly deep furrow, which is overlapped by the pendulous abdominal wall.

With the patient in the recumbent posture visible peristalsis is occasionally to be noted, if there is diastasis of the rectus muscles.

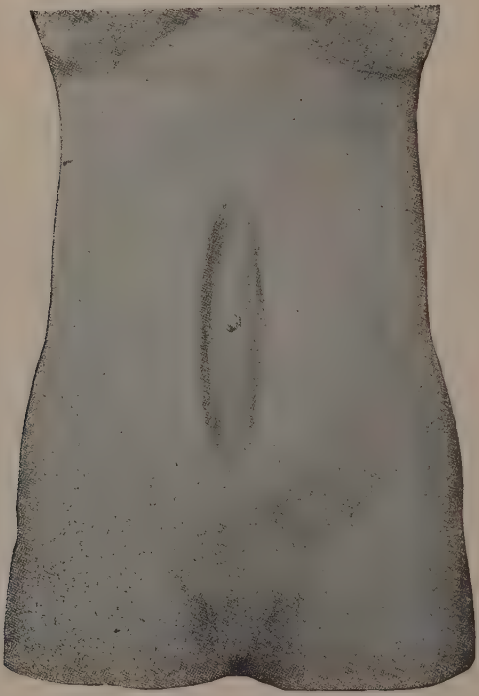


Fig. 85.—Median grooving of the abdominal wall where there is separation of the recti muscles. The woman is represented as lying on her back. (Crossen, after Webster.)

Asymmetrical Variations

Local bulging of the epigastric and hypochondriac regions of the abdomen may be significant of distention of the stomach, or enlargement of the liver, gall bladder, or spleen. Bulging of the abdominal wall involving the upper lateral regions of the abdomen and encroaching upon the umbilical region attends enlargements of the kidneys.

Undue prominence of the umbilical region alone occurs with umbilical hernia, and in the case of the progressive enlargement of a neoplasm of the intestine.

Local bulging of the abdominal wall confined to the hypogastric and iliac regions may be due to a distended urinary bladder, a pregnant uterus, uterine myoma, or ovarian cyst.

ABDOMINAL RETRACTION

In the presence of chronic wasting disease, during the course of prolonged diarrhea, and in the presence of stenosis of the cardiac or pyloric orifice of the stomach, the abdominal wall is retracted, the bony landmarks standing out very prominently. The abdomen appears to be ''scooped out'' like a boat, the scaphoid abdomen.

CHAPTER XIV

INSPECTION OF THE INGUINAL, GENITAL, AND PERINEAL REGIONS

INGUINAL REGION

Inspection of the inguinal region should include the condition of the glands, tumors, scars, and eruptions.

The glands in the groin are not visible in the normal subject, but are enlarged as a result of venereal infection or localized nonspecific inflammation resulting from abrasions about the external genitalia or of the lower extremities. Leucemia and metastases from malignant tumors of the genitals may also cause enlargement of the inguinal glands. The character of the adenopathy, whether hard and discrete, or soft and fluctuating with matting of the glands, should be determined.

A tumor in the inguinal region which protrudes when intra-abdominal pressure is increased, as by coughing, usually indicates a femoral or an inguinal hernia. Other inguinal tumors may result from abscesses or malignancy.

Scars may be the result of rupture or surgical evacuation of abscess, or from surgical repair of hernia.

Eruptions and erythema are common in obese individuals, due to irritation from the overhanging abdominal wall.

THE GENITALS

Male

The inspection of the male genitalia should begin with the distribution of the pubic hair. The normal distribution in the male extends upward for a varying distance along the midline of the abdomen, but not as far laterally as in the female. Parasites and parasite eggs are frequently found in the pubic hair.

The penis should be examined for developmental defects. All degrees of defect from a mere sulcus on the under side of the glans to a complete hypospadias may occur. In such cases, the urethral opening may occur anywhere along the inferior surface of the organ. Ulceration or injury may cause scarring, which may produce deformity.

Urethral discharge may be determined either by simple inspection of the urethral orifice or by stripping the urethra from base of the penis to the glans.

The prepuce should be retracted and carefully examined for scars or ulcerations. Congenital or acquired narrowing of the prepuce (phimosis) may prevent retraction and even narrow the urethral orifice.

The initial lesion of syphilis, chancre, often appears on the prepuce or glans as a round or oval, slightly elevated mass, often ulcerated and excreting a thin serous fluid. The irregular deep ulceration, chancroid, often emitting a foul odor, may be a secondary infected chancre and demands careful investigation.

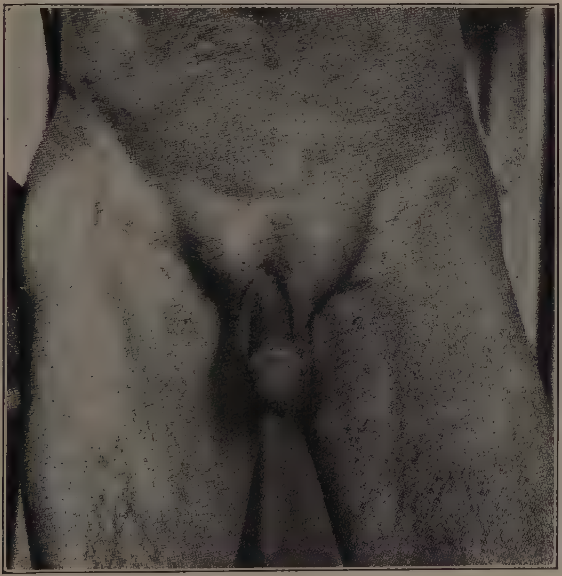


Fig. 86.—Bilateral direct inguinal hernia. (Watson.)

Inflammation of the glans (balanitis) may be accompanied by much swelling or edema. Malignancy of the penis may be associated with tumors and ulcerations. Simple herpes often occur on the penis, and in the early stages are often mistaken for chancre.

The scrotum should be examined for scars, eruptions, deformities, and enlarged veins. The epididymis is best examined by palpation, as are also the testicles and veins. Acute inflammations of the scrotum are accompanied by visible swelling and redness.

Fluid in the tunica vaginalis causes distention which can be readily diagnosed by transillumination with a pocket flash light (hydrocele). Inguinal hernia often descends into the scrotum and produces a distinct impulse when the patient coughs.

Visualization of the interior of the urethra and bladder with exploration of the ureters and kidneys is possible with the aid of a cystoscope and urethral catheters. The use of these instruments, however, should be demonstrated by a urologist in a course in urology. The x-ray is also a valuable aid to diagnosis of genitourinary conditions. See Chapter XXXIII, Radiology in Physical Diagnosis, page 483.

Female

The female genitalia are best inspected with the subject in the dorsal recumbent position, with the thighs flexed on the abdomen, and the knees flexed on the thighs. The knees should be widely separated.

Sufficient light is important. This may be supplied by a portable light or reflected from a head mirror. It is advisable to have an attendant present when making an examination of the female genitalia, both for the comfort of the patient and to forestall criticism or false accusations. The distribution of pubic hair should be noted as in the examination of the male genitals.

The mons veneris, labia major, and labia minor should be carefully inspected for deformities, scars, eruptions, and discharge. The clitoris, with prepuce, should be examined as to size, color, scars, eruptions, and abscesses. The opening of the urethra should be examined as to color, swelling, scars, and discharge. In the virgin, the color and shape of the hymen should be noted. (This membrane may be congenitally absent, and is not always absent even after copulation.)

The internal inspection of the vagina must be made with the aid of a speculum, but before the speculum is inserted it is wise to examine the interior of the vagina with the gloved finger, noting any irregularities or painful areas on the vaginal wall or cervix. The indiscriminate use of a speculum may provoke hemorrhage or produce unnecessary pain. (For details of vaginal palpation, see page 322.)

The vaginal speculum should be warm and thoroughly lubricated. It should then be inserted carefully, following the course of the vaginal canal by elevating or lowering its base. After insertion the speculum should be carefully opened to avoid pain or laceration.

The inspection should include the color of the vaginal mucous membrane, scars, vaginal discharge, the size of the cervix uteri, patency of the os, and evidence of laceration.

Normally, the mucous membrane of the vagina is of a pink color resembling the mucous membrane of the mouth, but in the presence of disease it may be bright scarlet or a deep bluish red color.

Scars, especially about the urethra and labia, may result from abscesses or ulcerations.

Vaginal discharge may vary in color from clear mucus to white, yellow, or greenish pus, and may be the result of gonorrheal infection or infection with pyogenic or saprophytic organisms.

The cervix uteri is often lacerated during childbirth, the os remaining large and frequently becoming chronically inflamed. Evidences of repair of lacerations should also be noted. Growths on the cervix may be irregular and warty in character. Carcinoma of the cervix is not uncommon.

THE PERINEUM

The perineum is a thick mass of muscular tissue lying between the genitals and the anus, and forming the pelvic floor. The skin is usually brownish in color. It should be examined for scars, eruptions and tumors. Its strength should be tested by instructing the patient to cough or strain.

Abscesses of the perineum are common in both sexes, and are often deep-seated, appearing as tumors of various sizes, which are exquisitely painful. Rupture of the perineal muscles is common during childbirth, the result varying from slight weakening of the pelvic floor to such complete destruction of muscle continuity as to allow bladder and rectum to protrude through the vaginal orifice. (For further details of gynecological examinations, the student is referred to textbooks and courses on gynecology.)

THE ANUS

Examination of the anus includes the tone of the external sphincter, tags, discharge, and tumors. Prolapse of rectum often occurs from weakening of the external sphincter or from cutting the sphincter when operating for the removal of hemorrhoids.

Inspection of the rectum requires a speculum. As in the case of the vaginal examination, a preliminary palpation with the gloved forefinger is necessary. (For details of rectal palpation, see page 323.) If masses or tender points are encountered, their cause should be determined if possible before inserting the speculum, or a smaller speculum should be used. The speculum should be warm and thoroughly lubricated. Insertion should be by gentle, firm pressure. Never apply force, as rupture of hemorrhoids or injury to the mucosa is all too common from careless use of this instrument. The mucous membrane should be carefully examined for color, tumors, scars, and ulcers. Enlarged veins often form tumor-like masses (hemorrhoids).

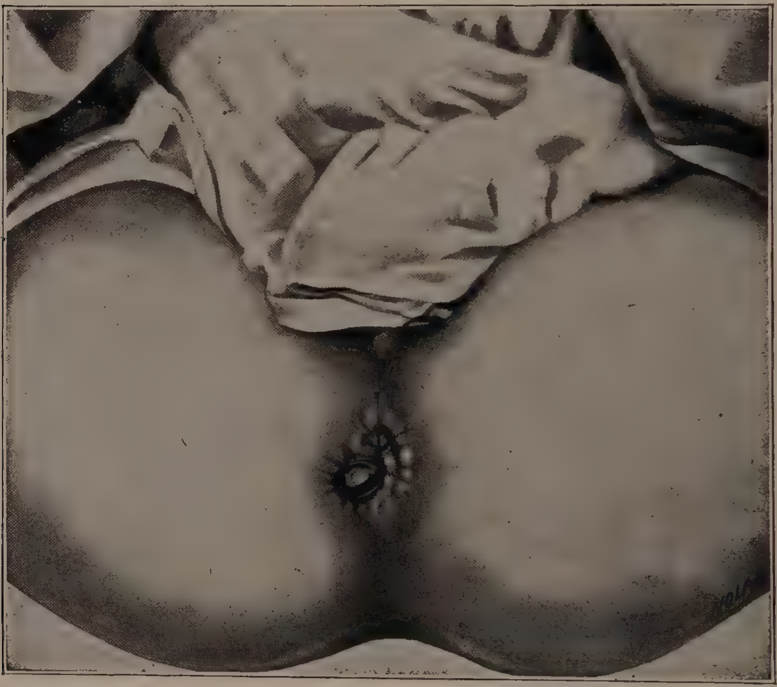


Fig. 87.—Acute external thrombotic hemorrhoid. (Hirschman.)

In the male, the prostate appears as a slight elevation on the anterior wall of the rectum, about 5 cm. above the external sphincter. It is often enlarged in men passed middle life and is subject to malignant disease.

In the female the uterus frequently presses on the anterior rectal wall and appears as an elongated tumor 3 to 4 cm. in length by 2 cm. in width. Retrodisplacement of the uterus may partially occlude the rectum. Irregular masses in the rectum may be due to carcinoma.

Abrasions of the rectal mucosa often become infected, causing fistulous tracts with one opening in the rectum and the other at a distant point. Bloody discharge may be the result of ruptured hemorrhoids or ulcerations.

CHAPTER XV

INSPECTION OF THE LOWER EXTREMITIES

When inspecting the lower extremities, careful comparison of the feet, legs, and thighs should be made. The feet should be examined for scars, eruptions, and deformities either congenital or acquired. All joints should be observed as to movement and enlargements. The subject should be instructed to stand on first one foot and then on the other, the examiner noting the plantar arches when under stress and when at rest. Pulsation of the dorsalis pedis artery should also be noted and checked by palpation.

THE TOES

Gangrene of the toes is usually significant of diabetes, arteriosclerosis, or Raynaud's disease. Gangrene of the toes is less frequently a sequence of frostbite, local trauma, ergotism, or embolism in connection with cardiac disease.

Perforating Ulcer.—The perforating ulcer, or mal perforante, occurring with locomotor ataxia and rarely with diabetes, is a deep circular ulcer, usually situated upon the under surface of the great toe.

Gout produces hot tense swelling of the metatarsophalangeal articulation of the great toe, which is very sensitive to pressure.

THE FOOT

Flat-foot, pes planus, is a flattening or giving way of the normal arch of the foot as a result of muscular paralysis, or ligamentous weakness from long standing or traumatism. Flat-foot is a sequence of rickets and infantile paralysis. Flat-foot is recognized by painting the sole of the foot with a colored fluid and causing the patient to stand upon a piece of paper, and noting whether an impression of the entire sole is left upon the paper.

Club-foot, or talipes, is a permanent fixation of the foot in deformity. In *talipes equinus* the heel is drawn up in such a manner that the patient walks upon the ball of the foot or the toes. In *talipes varus* the foot is inverted, the patient walking upon its outer border. In *talipes valgus* the foot is everted and the patient walks upon the inner border of the foot.

Enlargement of the foot with more or less distortion occurs in acromegaly, myxedema, and pulmonary osteoarthropathy.

Erythromelalgia.—In this condition the sole of the foot is very red and the seat of burning pain, which is made worse by walking and is relieved by elevating the limb.

Plantar warts are quite common and may cause considerable disability.

Absence of pulsation of the dorsalis pedis artery is often noted in diabetes and advanced arteriosclerosis.

The ankle joint should be tested by movements in all directions. Injuries and arthritis often produce stiff or immovable joints.

Paralysis of the extensor muscles of the leg is common in lead poisoning and produces the characteristic "foot drop." Recently many cases of paralysis of the extensor muscles of the leg have occurred from drinking Jamaica ginger.

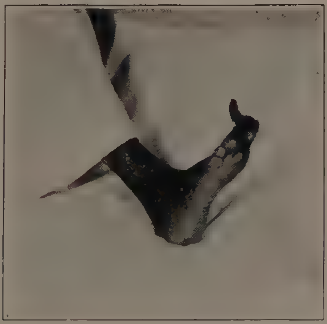


Fig. 88.—Gangrene of toes.

Edema of the lower extremities is first noted below the external malleolus. It may involve feet, legs and thighs.

THE LEG

The shape of the bones of the leg should be noted. The muscles should be compared as to size and symmetry. The skin should be examined for scars, eruptions and ulcerations. Superficial veins should be examined for shape, size and enlargements along their course.

Bowing of the tibiæ is most commonly due to rickets, but may also be noted in connection with osteitis deformans, mollities ossium, and cretinism.

Nodes.—Red, shining nodes situated over the tibiæ, which are very painful upon pressure are indicative of erythema nodosum, which is more frequently encountered here than in any other locality.

Deep nodular swellings, situated upon the tibia are due to syphilitic periostitis, while painless, noninflammatory indurated areas distributed over the leg may be gummata.

Leg Ulcers may be due to varicose veins, but are often due to tertiary syphilis, especially if there are multiple annular ulcers situated nearer the knee than the ankle.

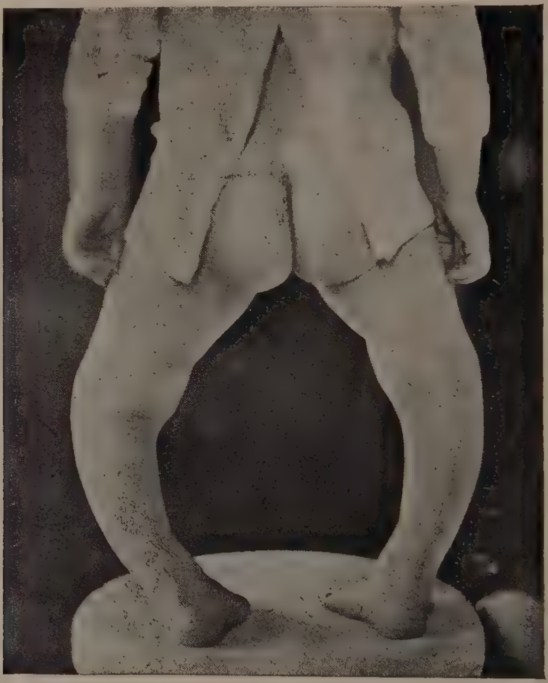


Fig. 89.—Showing extreme case of bowlegs. (From Woolley.)

Swelling of the Calves in children, associated with loss of muscular power and difficulty in rising to the erect posture, is indicative of pseudohypertrophic muscular palsy.

Atrophy of the muscles clothing the anterior and outer aspects of the leg is a sign of progressive muscular atrophy.

Varicose Veins.—Varicosities of the veins of the leg are indicative in some instances of prolonged standing, or the pressure of a pregnant uterus or tumor within the abdomen upon the vessels returning blood from the lower extremity.

The knee joints should be examined as to size and function. Arthritis of the knee joint may be caused by an acute rheumatic fever or by gonorrheal infection. The patella region should be examined for scars and enlargements; the popliteal space for skin eruptions and discolorations, usually due to superficial irritation. The blood vessels here are very superficial and are subject to injury, producing varicosities or aneurysm.

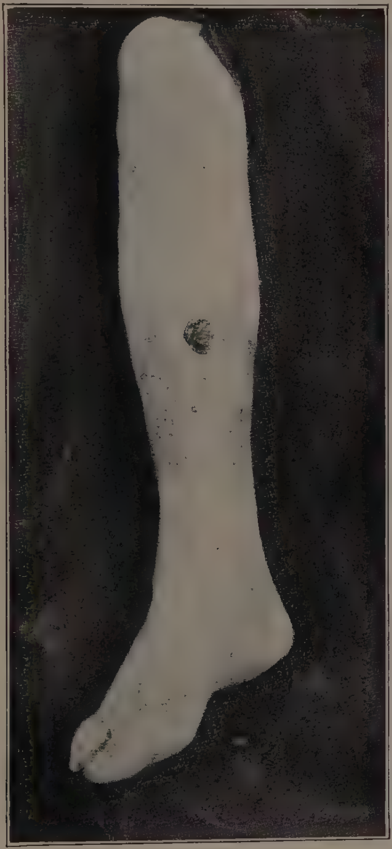


Fig. 90.—Varicose ulcer of leg. (From Eisendrath.)

Kernig's Sign.—In acute meningitis it is impossible to fully extend the leg upon the thigh. To elicit Kernig's Sign the patient should be placed upon the back with the thigh flexed at a right angle with the body. An effort is then made to extend the leg, bringing it in a line with the thigh. In the presence of meningitis it is difficult or impossible to extend the leg because of the marked flexor contracture of the hamstring muscles. In diagnosing meningitis by means of this sign it is necessary to exclude sciatica, old contractures, myositis, and tuberculous disease of the knee-joint.

Charcot's Joint.—In the course of locomotor ataxia not uncommonly as a result of trophic disturbance the knee joint assumes an enormous size, due to chronic inflammation of the synovial lining of the joint, which later progresses to the bone itself. The enlargement of the joint is always considerable and may become enormous. Early in the case the enlargement is due to effusion in the joint, but later it is produced by true osseous overgrowth. Pain is slight or is entirely absent. Usually affecting the knee-joint, the condition may involve the hip joint, and less commonly the ankle and the smaller articulations.

Housemaid's knee, produced by chronic bursitis of the prepatellar bursa as a consequence of persistent pressure upon the bursa incident to occupation, is characterized by effusion into the knee-joint, the effusion pushing the patella upward before it. Fluctuation can sometimes be obtained.

THE THIGHS

The thighs may vary slightly in the normal. The shape, condition of the skin, superficial veins, muscles, and movements of the hip joints should be observed and compared. The gluteal regions should be compared as to muscular development, scars and eruptions.

Edema of the thigh, affecting the leg and foot as well, possesses definite significance, depending upon whether it is unilateral or bilateral. Thus, edema of one lower extremity may result from varicose veins or thrombosis of the femoral vein. Bilateral edema points to cardiac insufficiency or hepatic disease producing general anasarca.

A chronic swelling of the lower end of the femur is often due to osteosarcoma of that bone.

Intermittent Claudication.—In subjects of arteriosclerosis an intermittent lameness may result from deficient circulation to the muscles of the thigh.

Swelling in Scarpa's Triangle.—A swelling in this portion of the thigh may be due to femoral hernia, or psoas abscess, the latter always occupying a position external to the femoral vessels.

Osteitis Deformans (Paget's Disease).—Osteitis deformans produces bowing of the bones of the thighs, with a consequent diminution of the stature. The head in this disease is characteristically deformed, and the contour of the thorax and abdomen is altered.

Osteomalacia, in its evolution is characterized by bowing of the bones of the lower extremity, produced by softening and rarefaction of the osseous structures peculiar to this disease.

Rickets.—In advanced rickets there is usually notable bowing of the bones of the lower extremities, leading to "bowlegs" or "knock-knees." In addition there are symmetrical swellings at the epiphyses of the long bones.

Pulmonary Osteoarthropathy.—In this disease the extremities of the long bones of the lower extremity, particularly of the tibiæ, participate in the chronic enlargement which characterizes the disease.

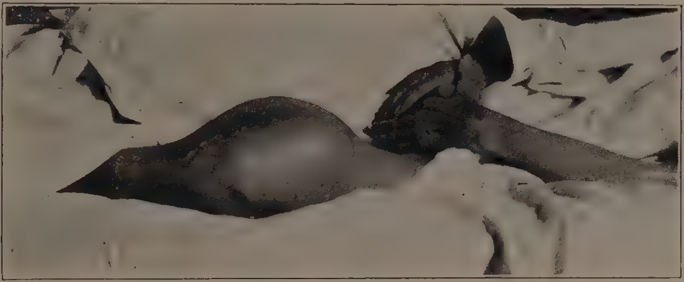


Fig. 91.—Osteosarcoma of femur.

Phlegmasia alba dolens, resulting from thrombosis of the femoral vein, produces swelling and edema of the thigh, with marked tenderness upon manipulation. The usual cause is puerperal sepsis, but this condition is also a not infrequent complication of typhoid fever.

Paralysis.—Paralysis of one leg if spastic is usually a part of a hemiplegia, but may rarely be due to a cortical lesion involving the leg center. Flaccid paralysis of one leg is the result of pressure neuritis, chronic lead poisoning, or anterior poliomyelitis.

Paralysis of both legs, paraplegia, may result from a cerebral lesion, as is the case in Little's disease, or may be due to transverse myelitis, disseminated sclerosis, or the late stages of locomotor ataxia.

SECTION IV

PALPATION

Palpation is the study of the various organs by the sense of touch. As with inspection, it requires thoroughness and systematic application. A special technic is required in palpating certain organs and regions, but on the whole more mistakes result from neglecting to palpate than by faulty technic. It is advisable to cultivate a light soothing touch and to avoid eliciting pain whenever possible, especially where the region to be palpated is suspected of being sensitive. As much information as possible should be acquired by the lightest possible touch before firmer palpation is attempted.

The hand of the examiner should be warm and sometimes lubricated by a little cocoa butter if the skin of the subject is dry.

CHAPTER XVI

PALPATION OF THE HEAD AND NECK

THE HEAD

Palpation of the vault of the cranium should be largely guided by inspection. For instance, if the region of the fontanels is depressed, elevated, or irregular, the condition should be checked by palpation. All scars should be carefully palpated for tenderness, elevation or depression, and any attachment to the deeper tissues.

A scar about the head that is freely movable is not likely to have been the cause of an injury sufficiently severe to produce injury to the bony vault.

Elevations about the head should be palpated for heat, tenderness, consistency, and mobility. Syphilitic nodes show no elevation of temperature, are not tender, feel hard, do not pit on pressure, and are not movable. Abscesses are hot, often exquisitely tender, are firm but not hard, often pit on pressure, and are somewhat movable. If free pus is present fluctuation may be made out by applying alternating pressure with the forefingers over the swelling.

The same plan should be followed in palpation of the forehead.

The Hair.—The hair should be palpated, noting whether it is soft or harsh, dry or oily, fine or coarse.

The Eyes.—The eyes should be especially palpated for ocular tension. This is best accomplished by directing the subject to look down, and gently palpating the eyeball with the index fingers placed upon the upper lid, making gentle pressure with first one and then the other, as in eliciting fluctuation. Comparing the "feel" with that of the other eye or with the eye of another person is often advisable. When there is suspicion of increased tension the opinion of a competent ophthalmologist should be secured. Increased tension is a prominent symptom of glaucoma. Diminished tension is found in degenerated eyeballs.

The Ears.—The ears should be palpated for irregularities of cartilage and for small lumps or tophi which appear between the skin and cartilage in gout. These tophi may or may not be visible.

The Mastoid Region.—The mastoid region should be palpated especially for tenderness. Inflammation of the mastoid cells often causes tenderness, heat, and a swelling, which pits on pressure.

The Face.—Palpation of the prominence of the malar bone may elicit pain when the maxillary sinuses are inflamed. Palpation of the nose, mouth and chin should be indicated by inspection, remembering the general rules as to prominences, depressions, and scars. The parotid region just below and anterior to the ear should be palpated for fluctuation as noted above. Scars should be palpated as noted above.

THE NECK

Palpation of the muscles, especially the sternomastoid, for tension and tenderness is often necessary, especially when a patient complains of pain or stiffness or when congenital or acquired spasm is noted. Palpation is best accomplished by placing the forefingers on each muscle and making gentle pressure and comparing the resistance.

The region anterior and posterior to the sternomastoid region is palpated for lymph glands. When present, they may be felt either with the forefinger or with the thumb and forefinger. When discrete they may be easily felt and their size, shape, consistency and tenderness noted. At times lymph glands may be matted together, forming a tumor of considerable size which may be palpated bimanually with the finger tips of both hands.

Glands in the submaxillary or occipital regions should be palpated in the same manner.

The Thyroid Gland.—When enlargement of the thyroid gland is visible it may be palpated with the thumb and finger of the right hand, noting size, consistency, tenderness, and pulsation. One should note whether one or both lobes or the isthmus is involved in the enlargement.

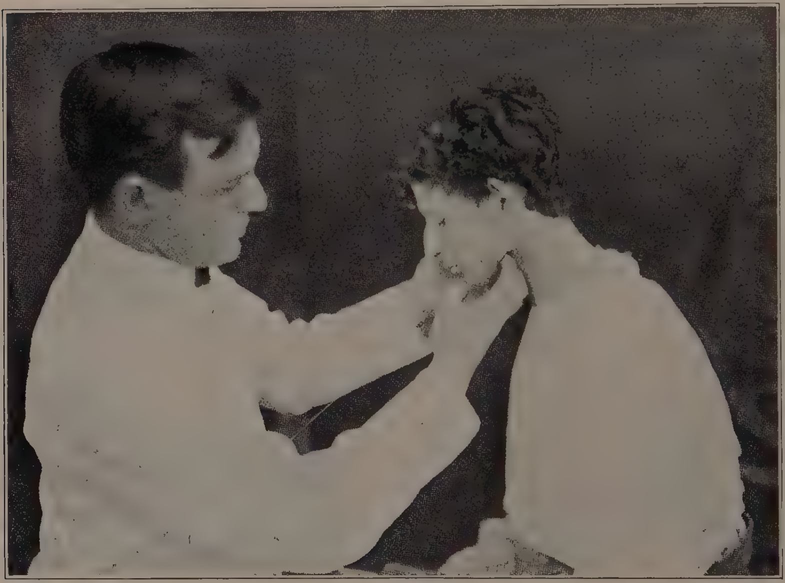


Fig. 92.—Palpation of submaxillary and submental glands. (From Eisendrath.)

The following method is recommended by Lahey. The thyroid cartilage is fixed by moderate pressure with the right thumb. The lobe on the opposite side is then palpated by hooking the left thumb behind the sternomastoid muscle and pressing forward the gland, which is then palpated with the fingers of the left hand. After palpation of one lobe the procedure is repeated on the opposite side.

The Carotid Artery.—Pulsations of the carotid artery can usually be palpated anterior to the lower third of the sternomastoid muscle. The size of the artery is noted, the rate and regularity both of time and force should be noted and compared with the artery on the opposite side. In aneurysm of the carotid artery

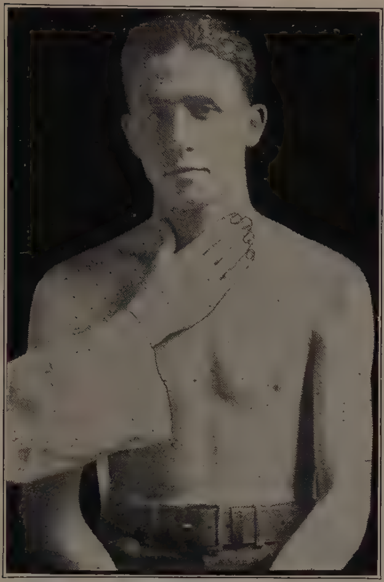


Fig. 93.—Palpation of thyroid gland.

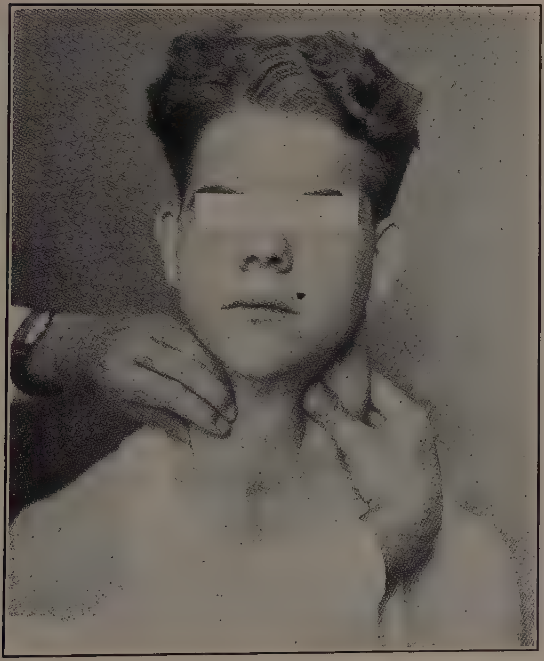


Fig. 94.—Lahey's method of palpating the thyroid gland.

there is at times a marked difference in force of the pulsation on the two sides. With aneurysm of the innominate artery the carotid pulse is often much weaker on the right side, while in aneurysm of the transverse portion of the arch of the aorta the left carotid pulse is often weaker than the right.

When there is a question of whether a vessel is an artery or vein, it can often be determined by making sufficient pressure to obliterate the vessel then stroking it upward. Arteries fill from below and veins from above. Venous valves can also be noted as small bulbous swellings on that portion of the vessel distal to the obstructing finger. Veins may sometimes be picked up, thus separating them from an underlying artery which transmits its pulsation to the vein. When such contact is removed the pulsation ceases.

Tumor Masses.—Tumor masses occurring in the neck should be palpated for shape, size, consistency, tenderness, and attachment. Branchial cysts noted elsewhere usually have a peculiar tense feel.

Trachea and Larynx.—In the presence of shifting of the mediastinal structures following collapse or extensive induration of one lung associated with compensatory emphysema of its fellow, palpation of the structures in the median line of the neck frequently shows a lateral displacement which is not visible to the eye. In palpating for minor grades of tracheal displacement, the examiner should place the palms upon the shoulders of the patient; and, gently insinuating the palmar aspect of the thumbs between the trachea and the sternomastoid muscle in the episternal region, search for a lateral deviation of the tube.

TRACHEAL TUG (OLIVER'S SIGN)

When adhesions have become established between an aortic aneurysm and the trachea or left bronchus, an appreciable descent of the trachea occurs with each pulsation of the aneurysmal sac. In eliciting Oliver's sign the examiner should stand behind the patient, who occupies the sitting posture with the lips closed and with the chin elevated in order to exert moderate traction upon the trachea through the cervical fascia. In this position the examiner should insert the tips of the thumb and index finger under the lower edge of the cricoid cartilage and exert gentle upward pressure upon these structures. In the presence of aneurysm of the aorta a systolic tug will be appreciated by the palpating digits.

In drawing conclusions from tracheal tugging, however, the examiner should bear in mind that while the sign is present with great constancy with aneurysm of the aortic arch, it has also been elicited in the presence of simple dilatation of the aorta; and in the presence of excessive hypertrophy of the heart a similar movement has been imparted to the trachea by strong pulsations in the innominate and carotid arteries.

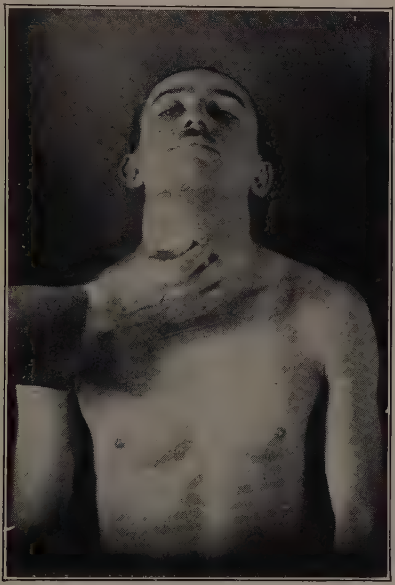


Fig. 95.—Elicitation of tracheal tug.

The Cervical Vertebræ.—The spines of the cervical vertebræ should be palpated with the thumb and forefinger to determine size, shape, and tenderness. While the fingers are thus placed, the subject should be instructed to move the head in various directions. Any roughness or grating during this maneuver may indicate disease of the vertebræ

In tuberculosis of the cervical vertebræ (Pott's disease), these vertebræ are often fixed, misshaped, and tender. More accurate information as to the condition of the cervical vertebræ is obtained from x-ray films.

CHAPTER XVII

PALPATION OF THE UPPER EXTREMITIES, POLYGRAPHY, AND SPHYGMOMANOMETRY

THE HAND AND WRIST

The hand should be palpated for temperature and moisture. The dry, cold hand, when not the result of exposure to low temperature, may mean fatigue or a weak heart. When only one hand is affected, it may mean partial occlusion of the brachial or radial artery, as in thrombosis. The cold moist hand is frequently noted in the asthenic individual, and in functional nervous disease. The warm moist hand is most common in hyperthyroidism but is, of course, common after exercise or exposure to high temperatures.

The muscular power of the hand can be tested roughly by having the subject grip the fingers of the examiner first with the right and then with the left hand. In right-handed individuals, the grip is usually stronger in the right hand, and vice versa. Special training of muscles, as in operating machines, may cause greater development of one hand, and one may thus erroneously consider the less developed hand the diseased one.

Weakness of muscles may be due to central nervous disease, such as cerebral hemorrhage or embolism, in which the degree of muscular weakness may vary from paralysis to scarcely demonstrable weakness. Peripheral neuritis may also cause varying degrees of weakness, as may injury to brachial plexus or peripheral nerves.

Palpation may also be used in the study of muscular spasms, as noted on inspection. Tumors of the hands should also be checked by palpation. Joint movements may be palpated for roughness or grating sensations. Such sensations are noted in certain types of arthritis.

Palpation of the wrist joint may also reveal roughness or grating sensation due to arthritis. X-ray examination should be used to confirm such observations.

The Pulse

The radial artery usually can be felt as it passes over the head of the radius, but occasionally it divides or passes over the lateral aspect of the bone and is most easily felt outside the thumb tendon in the "anatomical snuff box." This anomaly may occur on one or both sides. Occasionally the artery is beneath the tendons and cannot be felt.

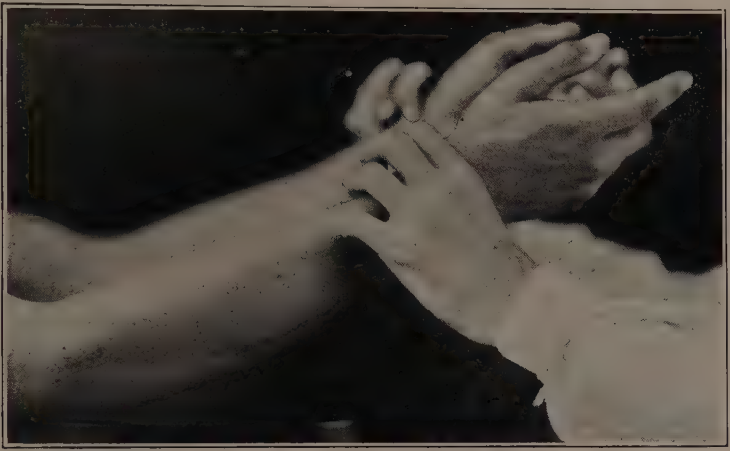


Fig. 96.—Palpation of the radial pulses.

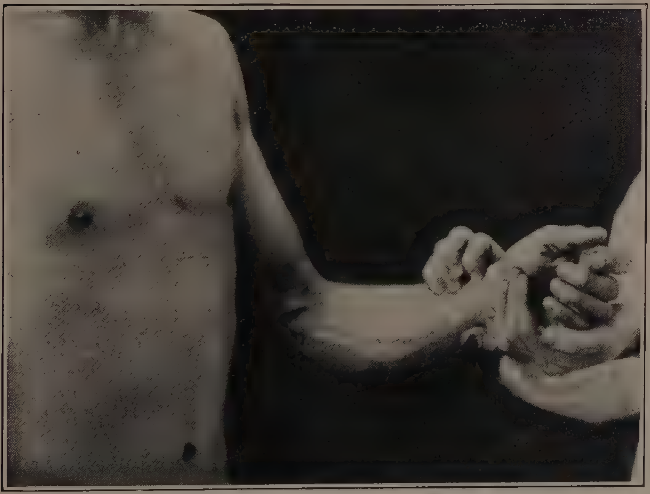


Fig. 97.—Palpation of the radial pulse.

The palpation of the radial pulse gives us valuable information as to the condition of the circulatory apparatus.

During palpation of the radial pulse the patient should occupy the sitting or recumbent posture, with the arm extended upon a level with the heart. The examiner seated beside the patient should first apply the tips of the first three fingers over the course of the radial artery.

The examiner should never reach around the wrist nor should the thumb be used in examining a pulse. In the former, sensation is distributed over too large a surface and is likely to distract the examiner's attention. In the latter, the artery in the thumb of the examiner also produces a pulsation which is confusing. Both radial arteries should be palpated at the same time, especially when the subject is examined for the first time.

Bilateral Symmetry of the Pulses.—In the normal subject there is no appreciable difference in the radial pulse as recorded at the two wrists. This symmetry of the pulses may, however, be disturbed even to the total absence of the pulse at one wrist. Aneurysm of the ascending aorta or innominate artery may retard the right radial pulse; while aneurysm of the subclavian, axillary or brachial artery may cause retardation upon either side of the body.

Fracture of the bones of the arm or injuries producing cicatricial compression of the artery in the axilla or arm, as well as compression by tumors or enlarged glands, will alter the character of the pulse at the wrist and cause asymmetry of the pulses. Pneumothorax or massive pleural effusion, by compression of the subclavian artery, may cause a retardation or may alter the character of one radial pulse.

The examiner should pass the finger tips longitudinally along the course of the artery and transversely across the artery, and, while rolling the vessel beneath the fingers, study the several factors which enter into the analysis of the pulse. When the radial artery is so palpated it is observed that at each pulse wave the artery is changed from a flat tube into a circular one, and moreover, that the vessel lengthens or straightens appreciably during this period.

Rate.—It is desirable in all cases to count the pulse for a full minute in recording the rate of the pulse. The pulse may, however, be counted during twenty seconds and the result multiplied by three; or it may be counted during thirty seconds and the result multiplied by two. When the pulse is so rapid that it is impossible to count all of the individual beats, an approximate estimate of the frequency of the pulse may be made by endeavor-ing to count every other beat, or the examiner may make dots with a pencil held in the unengaged hand and count the number of dots which are made during a minute.

The normal pulse rate in the adult subject varies from 70 to 80 beats per minute. The rate of the pulse, however, is modified by physiologic factors, such as the age and sex of the subject, the size of the body, the position assumed by the subject, and the relation of the time of recording the pulse to the ingestion of food.

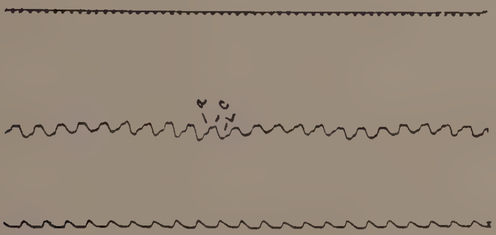


Fig. 98.—Pulsus frequens.

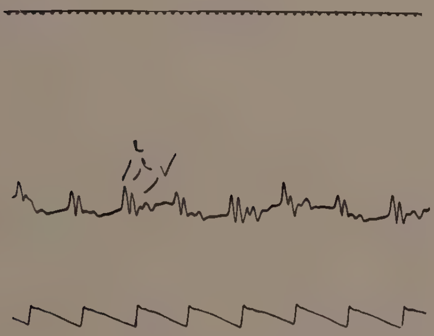


Fig. 99.—Pulsus rarus.

At birth the pulse rate is approximately 130 to 140 beats per minute; during the first year of life it is 115 to 130; at the seventh year it averages 85 to 90; while in the aged subject it drops to 60 to 70 beats per minute. In women the pulse is usually moderately increased in frequency over the rate obtaining in the adult male subject.

The size of the body exerts a slight influence upon the rate of the pulse, the rate being lower in large subjects than it is in persons of small stature. The position of the patient influences the pulse rate, the pulse being constantly more rapid when counted in the upright posture than when it is taken with the patient in the recumbent position. The rate is likewise quickened for an hour or two following a full meal. Exercise and mental or emotional excitement serve to cause a transient increase in the pulse rate, not infrequently doubling the rate for the individual. Finally, the ingestion of excessive quantities of tea or coffee, or the excessive use of tobacco may be responsible for a transient elevation of the pulse rate.

Pathologically, elevation of the pulse rate, evinced by the *pulsus frequens,* is noted in disturbances of the innervation of the heart, stimulation of the sympathetic innervation from thoracic or abdominal disease and inhibition of the vagus innervation resulting in reflex tachycardia. Increase in the pulse frequency attends the majority of febrile diseases, the pulse rate being increased approximately eight beats for each degree of pyrexia. Exceptions to this rule are the febrile elevations of yellow fever, typhoid fever, and tuberculous meningitis, in which the pulse rate may actually exhibit a retardation.

In the presence of paroxysmal tachycardia the frequency of the pulse may be so great that it is impossible to count the radial pulse. In this instance the tachycardia is usually of abrupt onset, of variable duration, and it commonly ceases as abruptly as it began. The rhythm of the pulse during the paroxysm is usually regular, but arrhythmia at the onset and conclusion of the attack is common. Similarly, in auricular flutter and fibrillation a rapid rate is the rule, frequently with disturbances in the rhythm of the pulse.

Diminution in the pulse rate, reflecting a state of bradycardia and evinced by the presence of the *pulsus rarus,* in which the pulsations fall below 60 beats per minute, occurs in a physiologic and a pathologic form.

Physiologic diminution of the pulse rate is a constant accompaniment of advanced age, whereas in other instances it is an inherent physical characteristic. During the puerperal state and during parturition the pulse is commonly slowed without possessing untoward significance. A similar slowing of the pulse is noted during hunger and exhaustion from any cause. Strictly speaking, the persistent pulsus rarus which is often noted during convalescence from febrile diseases in the case of young robust subjects is physiologic and is an evidence of exhaustion of the patient.

Pathologic slowing of the pulse accompanies increased intracranial tension in meningitis, cerebral hemorrhage, abscess or tumor, and depressed fracture of the skull. In cardiovascular disease a slow pulse is frequently noted during aortic stenosis, coronary sclerosis, and Stokes-Adams' disease. In disease of the urinary system, the pulsus rarus is noted during the paroxysm of renal colic and in uremia. It likewise attends gall-stone colic, autointoxication, and toxic jaundice. In chronic lead poisoning and in intoxication from alcohol, opium or digitalis the pulse rate is frequently slowed to a remarkable degree.

Diminution of the pulse rate may be due to irritation of the trunk of the vagus nerve by mediastinal tumor, enlarged mediastinal glands, aortic aneurysm or extensive empyema. Similarly, depression of the sympathetic innervation, with consequent inhibition of the accelerator impulses to the myocardium, results in slowing of the pulse.

Condition of the Arterial Wall.—In studying the condition of the wall of the radial artery the examiner should endeavor to exclude the blood from the artery by compression with the index and third fingers, in the meantime rolling the artery between the middle finger and the subjacent radius. During this maneuver counterpressure should be applied to the dorsum of the wrist with the thumb. The wall of the normal radial artery is soft and yielding, and is to be distinguished with difficulty from the surrounding tissues. Any departure from the normal elastic state of the artery is of diagnostic significance; but in this connection the examiner should not mistake increased tension of the pulse for thickening of the arterial wall.

In the aged subject and in the younger subject in the presence of arteriosclerosis due to alcoholism, syphilis, gout, or other cause, the arterial wall presents varying grades of resistance to manual compression. In advanced stages of arterial fibrosis the artery may frequently be rolled between the fingers and the lower end of the radius, yielding a tactile impression similar to palpation of a hard reed or pipe stem. It is to be borne in mind, however, that the detection of sclerosis of the radial artery does not necessarily signify a similar grade of hardening of the aorta and coronary arteries. In other instances the artery is beaded and more or less tortuous, owing to the presence of atheromatous plaques or beads in the arterial wall.

The Size of the Artery.—The radial artery may be larger or smaller than normal as a result of congenital deformity, without possessing untoward significance. Transient changes in the size of the arterial pulsation, dependent upon changes in the myocardium and disturbances in the vasomotor system, are noted in the section dealing with the volume of the pulse.

Rhythm.—In the normal subject the several pulse beats are of uniform force, and the beats are separated by equal intervals. It follows, therefore, that a disturbance in the rhythm of the pulse may have reference either to the force or to the temporal sequence of the beats.

Arrhythmia of the pulse is betrayed by a deviation from the normal orderly sequence of the beats, with or without the omission of beats. The disorder is attributable to variations in vagus tone, to lesions which interfere with the conduction of the contraction impulse from auricle to ventricle, or to the generation of impulses to contraction in ectopic foci in the auricle, ventricle, or auriculoventricular node and bundle. The characteristic type which the arrhythmia assumes usually furnishes a reliable clue to the nature of the lesion which underlies the disturbance.

Intermission is the occasional or persistent omission of a radial pulse beat. Depending upon the underlying cause, the omission may occur at irregular intervals, or it may follow an orderly sequence with the consequent production of a characteristic rhythm. Frequently polygraphic records or electrocardiograms are necessary in the elucidation of such a rhythm.

In the analysis of a case of intermission it is important to determine by auscultation of the precordia whether the omission of the radial pulse is due to an absence of ventricular systole, caused by depression of the auriculoventricular bundle, the *pulsus deficiens;* or whether it is due to a premature ventricular contraction which is too feeble to open the aortic valve, the *pulsus intermittens.*

When the omissions of the pulse beats follow a regular sequence, when they are rhythmically irregular, the result is the production of an *allorhythmic pulse.* In this variety of intermission belong the *pulsus bigeminus,* in which two beats occur in regular sequence and are followed by an omission, and the *pulsus trigeminus,* in which three beats occur regularly, and are followed by an omission of the pulse. Bigeminy of the pulse is caused by premature contractions occurring regularly after every second beat but failing to reach the wrist, by the occurrence of a premature contraction reaching the wrist after every normal beat, or by 3:2 heart-block. Trigeminy of the pulse is noted when in partial heart-block every fourth auricular impulse fails to reach the ventricle.

In the presence of isolated premature contractions of the ventricle, the *pulsus intercidens* is noted, in which, following a sequence of regular beats, the last beat is quickly followed by a minor pulse wave.

Pulsus paradoxus is a pulse in which toward the completion of inspiration the beats become feeble, and at times become imperceptible at the wrist. This type of pulse was first described by Griesinger in 1854 and was further studied by Kussmaul in 1873. The precise mode of generation of this abnormality of the pulse has not been adequately explained. Müller and Wenckebach noted that the effort to inspire deeply with the glottis closed was attended by a similar alteration in the radial pulse; but Griesinger and Kussmaul pointed out that the clinical pulsus paradoxus is in evidence during tranquil respiration, and hence has

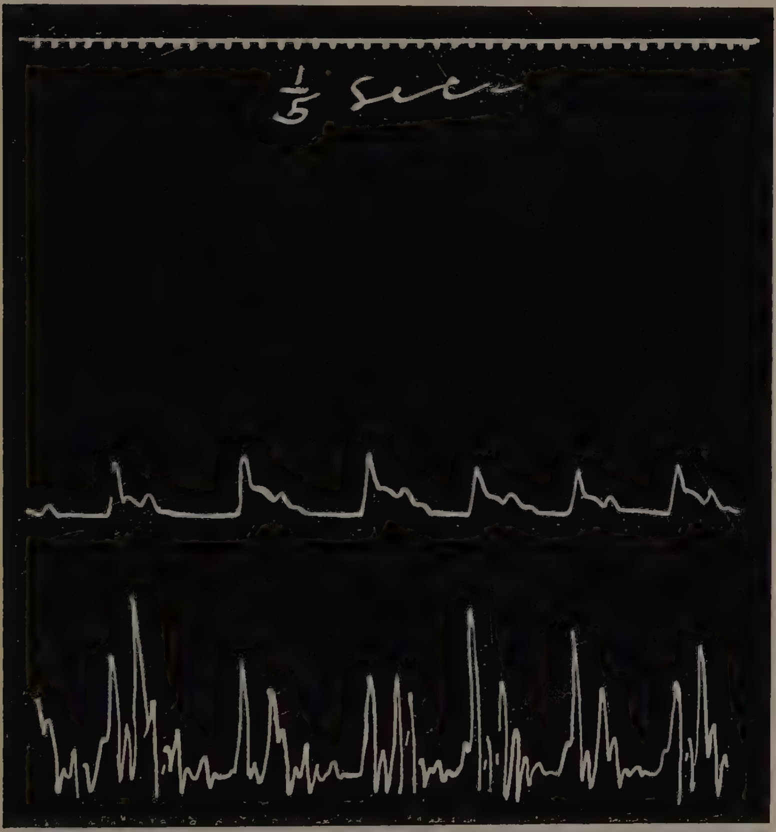


Fig. 100.—Pulsus bigeminus.

nothing in common with the experiment of Müller and Wenckebach.

Kussmaul held that pulsus paradoxus is produced by adhesions between the aorta and sternum, and that the traction of these bands upon the vessel during the inspiratory expansion of the thorax results in a narrowing of the aorta. Svoiechotow has noted the pulse in chronic adhesive pericarditis in which ad-

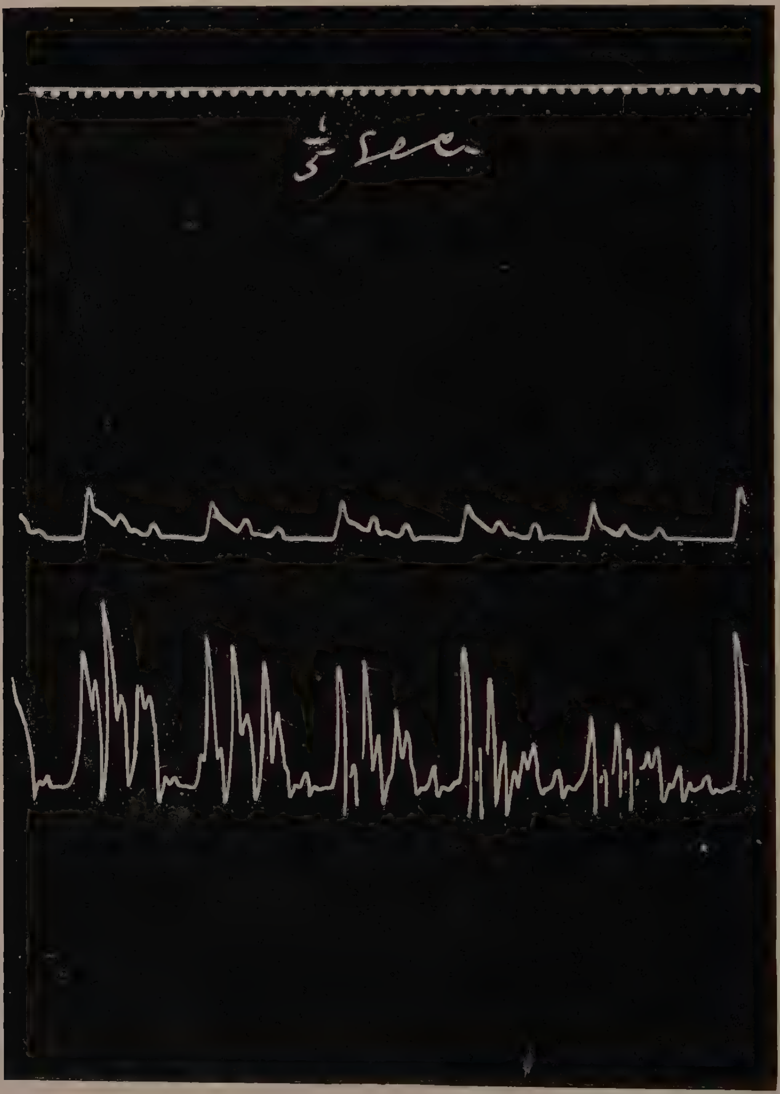


Fig. 101.—Pulsus trigeminus.

hesions united the pericardium to the pleura and left lung. The paradoxical pulse has likewise been noted during the inspiratory dyspnea of laryngismus stridulus, in serofibrinous pericarditis with effusion, and in connection with mediastinal inflammation and neoplasm.

The antithesis of the paradoxical pulse is occasionally observed, in which the radial pulse trails off and becomes imperceptible during expiration (Riegel's pulse).

Diminution in the force of alternate pulse beats is encountered in the *pulsus alternans,* with or without disturbance in the temporal rhythm. Since its recognition by Traube in 1872, various hypotheses have been brought forward in the effort to explain the pathogenesis of this peculiar rhythm. The theory of Wenckebach holds that the alternation is produced by regularly recurring variations in the contractility or irritability of the myocardium, with the result that the ventricles contract to a maximum degree only during alternate beats. On the contrary, Hering and Gaskell attribute the abnormal rhythm to alternating hyposystole occasioned by a restriction of the areas of the myocardium which pass into contraction. While the pathogenesis of the rhythm is not altogether clear, clinical experience indicates that the disturbance is of grave prognostic significance, pointing to a varying grade of degeneration of the myocardium. The rhythm is encountered most frequently during late stages of chronic myocarditis, arteriosclerosis, and chronic nephritis.

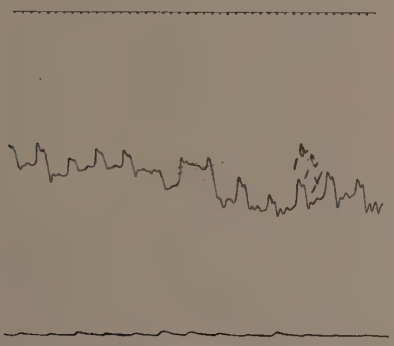


Fig. 102.—Paradoxical pulse.

Volume.—The volume or size of the pulse is dependent upon the size of the artery, the amount of blood which is expelled during ventricular systole, the ability of the aortic valve to prevent regurgitation of the blood column, and upon the state of the vasomotor nervous system. Thus, in the presence of powerful ventricular contractions associated with vasomotor depression during the course of acute febrile diseases the radial pulse is full and bounding, constituting the *pulsus magnus.* Similarly, hypertrophy of the left ventricle with competence of the aortic valve is attended by the pulsus magnus.

When the artery is palpably filled with blood in the intervals between the individual pulse beats, indicating the presence of a large bulk of circulating blood, the pulse is termed the *pulsus plenus.*

In the presence of cardiac asthenia, or when as a result of regurgitant or stenotic lesions at the aortic or mitral valves a diminished quota of blood is expelled from the ventricle during systole, the radial pulse is of small volume, the *pulsus parvus.* A similar pulse is noted in the presence of extreme grades of anemia as a result of diminution in the total bulk of the circulating blood.

When the artery is palpably empty and readily compressible during the intervals between the pulse beats, indicating a diminution in the bulk of the circulating blood, the pulse is termed the *pulsus vacuus.*

Force.—The force of the pulse depends upon the energy with which the left ventricle contracts and upon the elasticity of the arterial walls. If the ventricle is hypertrophied but the arterial walls have lost their elasticity as a result of fibrosis, much of the force of the heart consumed in the expulsion of the blood is lost through the absence of the elastic recoil of the arteries.

In general, the force of the pulse is increased in conditions attended by cardiac hypertrophy, and it is diminished in the presence of cardiac asthenia and dilatation. Moderate stimulation of the vagus nerves increases the pulse force by slowing the heart and increasing its period of repose; but if the stimulation is extreme, the ventricular contractions are so few that the diminished blood content of the arterial system is not sufficient for the powerful ventricular systole to act upon, and the force of the pulse is consequently diminished.

Tension.—The tension of the pulse is influenced by the power and the rate of ventricular contraction, the volume of the circulating blood, the elasticity of the arterial walls, and the degree of peripheral resistance to the blood flow. In the presence of arteriosclerosis and chronic renal disease associated with hypertrophy of the left ventricle, arterial tension is raised (hypertension), as evinced by a hard pulse, which is compressed with distinct effort, the *pulsus durus.* Transient angiospasm is attended by a similar pulse of high tension, but the alteration in tension in this instance is subject to fluctuations and is not permanent.

When, on the other hand, the output of blood from the ventricle is diminished by cardiac dilatation or valvular disease, combined with vasodilatation or a diminution in the amount of circulating blood as a result of hemorrhage, anemia or cachexia, the arterial pressure is diminished (hypotension), and the pulse is soft and readily compressible, the *pulsus mollis.*

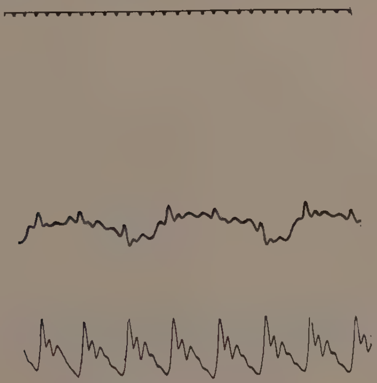


Fig. 103.—Pulsus magnus.

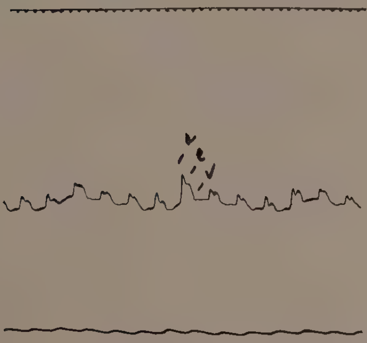


Fig. 104.—Pulsus parvus.

Size and Shape of the Wave.—The size and shape of the pulse wave is determined by the duration of the ventricular systole, the recoil of the artery and the degree of peripheral resistance.

The duration of the pulse is increased in the slow, sluggish *pulsus tardus,* which is always associated with increased peripheral resistance due to constriction of the small arterioles, such as occurs in arteriosclerosis, chronic renal disease, and angina pectoris. The

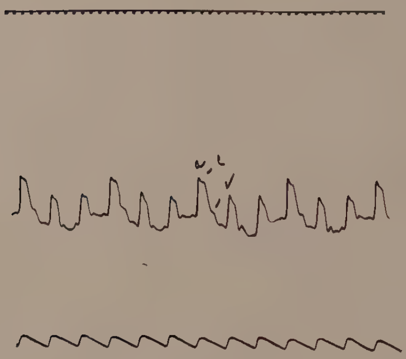


Fig. 105.—Pulsus tardus.

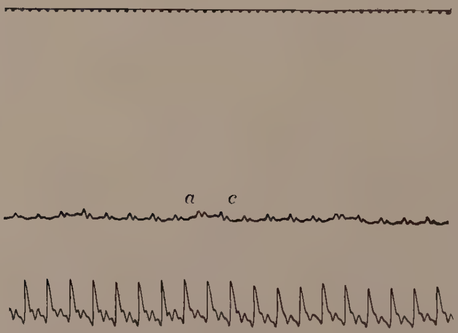


Fig. 106.—Water-hammer pulse (pulsus celer).

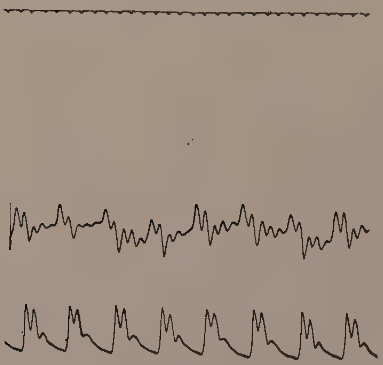


Fig. 107.—Pulsus bisferiens.

sphygmogram of this pulse shows a gradual up-stroke, a well sustained apex or plateau, and a gradual fall to the base line. The pulse is likewise observed in aortic stenosis, in which case it is to be ascribed to the prolonged systole of the left ventricle.

The duration of the pulse is diminished with the production of a very brief pulse wave, the *pulsus celer,* in conditions associated with diminished peripheral resistance as a result of relaxation of the arterioles in the course of acute fevers. The pulse is also encountered in the presence of aortic regurgitation, constituting the *water-hammer* or *Corrigan pulse* of this disease. The pulse is characterized by a sudden and full expansion of the artery, followed by a sudden collapse of the vessel under the fingers. The pulse is readily demonstrated by grasping the wrist and elevating the arm above the level of the heart. The sphygmogram of the pulsus celer shows an abrupt up-stroke, followed by an acute angle and by a rapid fall of the down-stroke to the base line.

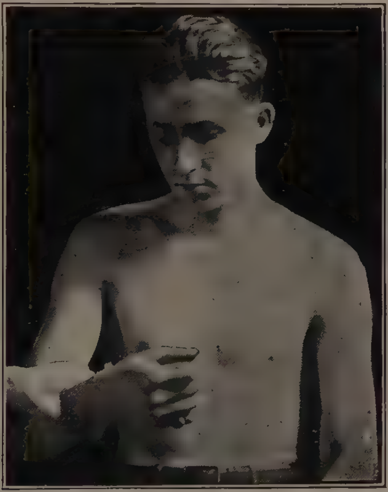


Fig. 108.—Method of detection of water-hammer pulse.

Occasionally during the course of a continuous fever associated with vasodilatation, there is noted a pulse of low tension and diminished rate but of full volume, in which there is a reduplication appreciable to the palpating fingers as a minor beat, following the principal pulse beat, the *dicrotic pulse.* The dicrotic palpable wave is to be attributed to excessive elasticitiy of the arteries combined with general relaxation of the smaller arterioles. The pulse is occasionally demonstrable in the presence of imperfectly compensated mitral regurgitation, during typhoid fever, and in states of anemia and exhaustion.

Very rarely in extreme grades of aortic stenosis there is a palpable tidal wave of the artery, following immediately upon the principal beat, corresponding to the tidal wave of the sphygmogram, constituting the *pulsus bisferiens.* The pulse is distinguished from the dicrotic pulse by the fact that it is encountered in the presence of arterial hypertension, and the second wave is appreciable immediately subsequent to the principal pulse beat.

The Capillary Pulse

While capillary pulsation is a procedure connected with inspection, it is best studied along with other pulse changes.

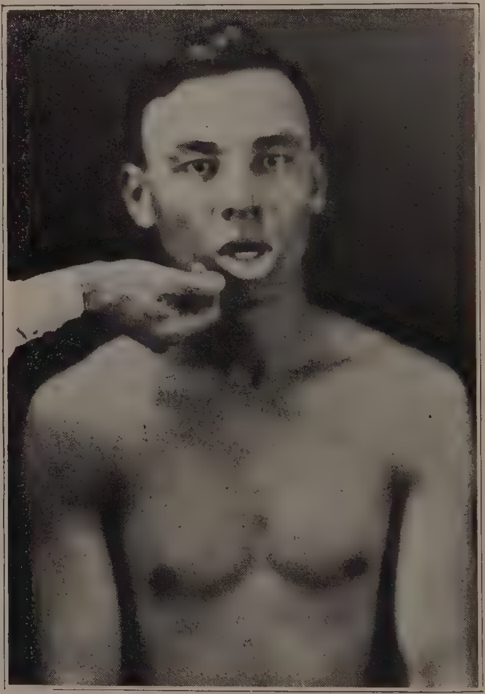


Fig. 109.—Demonstration of capillary pulse.

Systolic pulsation in the capillaries is sometimes a normal phenomenon; it may be the result of temporary loss of vasomotor tone during anemia or febrile diseases; but it is also a very valuable sign of aortic regurgitation or Corrigan's disease.

There are several methods of demonstrating capillary pulsation. A serviceable method is by blanching the finger nail by the exertion of moderate pressure upon the tip of the nail, whereupon a systolic flushing and a diastolic blanching of the subungual tissues will be observed. Another method of demonstrating the phenomenon is by drawing the nail over the forehead and producing a line, which is alternately flushed and blanched during ventricular systole and diastole. A third method of detecting capillary pulsation is by covering the lower lip by a glass slide and observing the systolic flushing and diastolic blanching of the lip which is compressed by the slide.

The capillary pulse is frequently accompanied by visible pulsation in the carotid arteries, and by pulsation in the veins of the dorsum of the hand or foot.

The Centripetal Venous Pulse

A visible pulsation, the centripetal or penetrating venous pulse, is occasionally visible in the veins of the dorsum of the hand or foot, or in the delicate mammary veins. This pulsation is most frequently associated with aortic regurgitation, and less commonly with anemia, constituting in the first instance an exaggeration of the capillary pulse of Quincke.

POLYGRAPHY

With the clinical polygraph of Jaquet or Mackenzie, synchronous tracings may be obtained of the radial pulse, the cardiac impulse, and a third pulsating area, whether it be the carotid artery, jugular vein, or hepatic pulsation. Under these circumstances the sphygmogram obtained from the carotid or radial artery yields information relative to the movements of the left side of the heart; the cardiogram, recorded over the cardiac impulse, records the mechanical displacement of the heart against the thoracic wall; and the jugular tracing yields information pertaining to venous pressure and the movements of the right side of the heart.

The Sphygmogram.—The sphygmogram portrays the rate and rhythm of the heart beat, and it is of material assistance in the analysis of the jugular tracing, which taken as an isolated tracing, would be devoid of diagnostic significance.

The normal sphygmogram consists of an almost vertical upstroke, termed the percussion stroke or anacrotic limb, of an apex or angle, and of a down-stroke or catacrotic limb. The percussion stroke is produced by the impact of the pulse wave against the pad of the sphygmograph; and, in the case of the radial artery, occurs approximately 0.1 second subsequent to the opening of the semilunar valves.

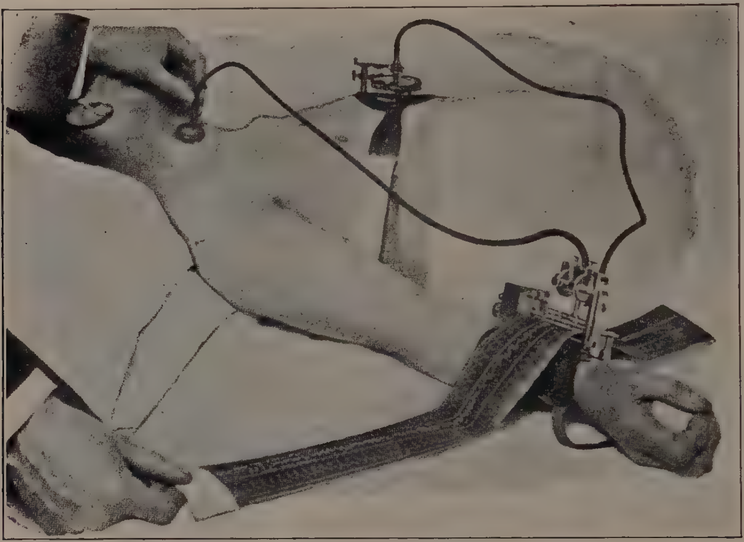


Fig. 110.—Jaquet sphygmocardiograph. Simultaneous tracings of radial pulse, cardiac impulse, and carotid pulse.

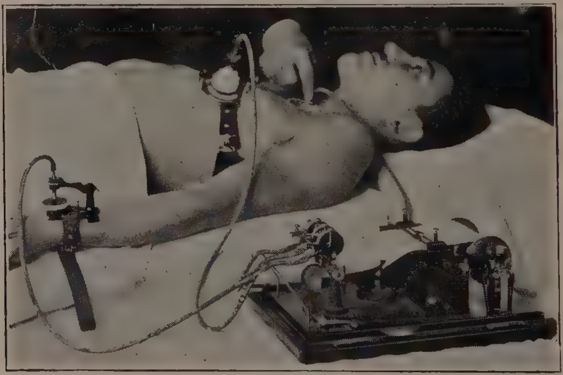


Fig. 111.—MacKenzie Ink Polygraph, American Model.

In the typical tracing the catacrotic limb presents two waves, the *predicrotic* or *tidal* wave, and the *dicrotic* wave. The dicrotic wave is preceded by a notch, the *dicrotic* or *aortic* notch, indicating the point at which the aortic valve closes. The sphygmic period is embraced between the commencement of the percussion stroke and the dicrotic notch.

The *apex* of the sphymogram varies in shape with the type of instrument employed and with the refinement of the technic of the operator. Its shape is in no wise constant, and it possesses no diagnostic or prognostic significance.

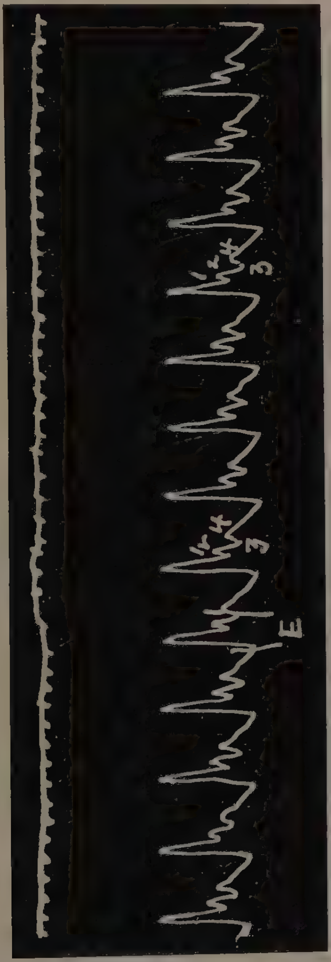


Fig. 112.—Normal sphygmogram. *1*, Predicrotic notch; *2*, tidal wave; *3*, dicrotic notch; *4*, dicrotic wave; *E*, sphygmic period.

The *cardiogram,* the tracing recorded over the cardiac impulse, simply records the impact of the cardiac apex against the thoracic wall. It yields no information as to the intraventricular pressure.

The normal cardiogram presents an almost vertical upstroke, coinciding with the commencement of ventricular systole, followed by a sustained apex, the "systolic plateau," which in turn is followed by an abrupt downstroke, as the heart falls backward from the chest wall.

The commencement of the upstroke of the cardiogram corresponds to the earliest portion of ventricular systole, during which the intraventricular pressure is rapidly rising, while the semilunar valves are as yet closed, the presphygmic period. Comparison of the cardiogram with a simultaneous carotid tracing will show that the semilunar valves open at the commencement of the systolic plateau, thus ushering in the sphygmic period of the cardiac cycle. At the termination of the plateau, which corresponds to the dicrotic notch of the carotid tracing, the semilunar valves close, and the sphygmic period terminates. Thus it is observed that the duration of the sphygmic period may be

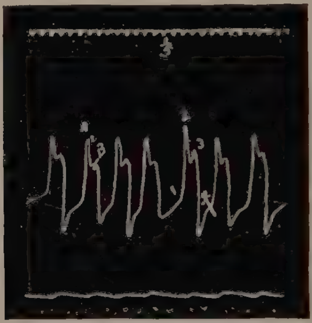
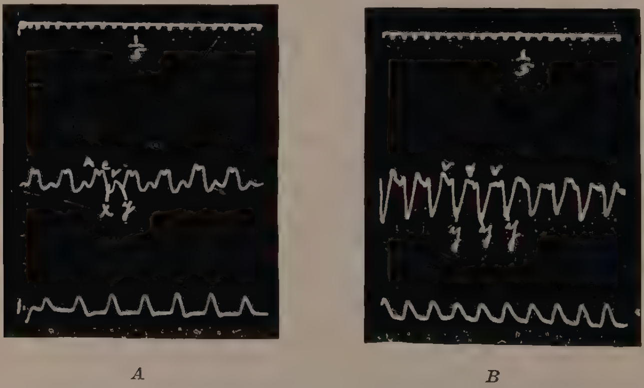


Fig. 113.—Normal cardiogram. *A*, Semilunar valves open; *B*, semilunar valves closed; *A-B*, sphygmic period; *1-2-3*, duration of ventricular systole; *3-4*, ventricular diastole.



A *B*

Fig. 114-*A*.—Auricular type of venous pulse. *A*, auricular wave; *C*, carotid wave; *V*, ventricular wave; *X*, first negative phase. *y*, second negative phase.

Fig. 114-*B*.—Ventricular type of venous pulse. *V*, ventricular wave; *y*, second negative phase.

estimated from the cardiogram, and that the events portrayed in this tracing may be utilized in the analysis of the venous pulse.

The Venous Pulse.—The normal *phlebogram* recorded over the bulb of the jugular vein, representing the physiologic, auricular or negative venous pulse, consists of three waves, which are dependent upon movements of the right side of the heart for their generation.

The first wave, the *auricular,* or *a-wave,* corresponds to auricular systole and is produced by the sudden arrest of the venous flow incident to systole of the right auricle. Under normal conditions the venous orifices of the right auricle are closed during auricular systole and the venous flow is interrupted, with the production of a normal a-wave. When the right heart is engorged, however, there is regurgitation of auricular blood during systole, with the production of an unduly large a-wave. Similarly, in the infrequent cases of nodal rhythm, when the auricles and ventricles contract synchronously so that the blood cannot be expelled from the auricle into the ventricle, the a-wave possesses an increased magnitude.

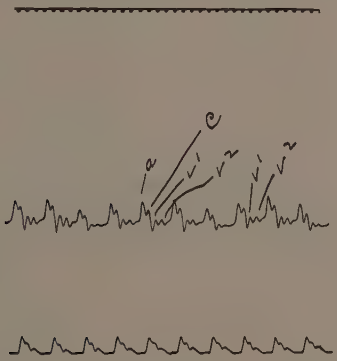


Fig. 115.—Auricular venous pulse. *V-1* and *V-2* are well shown.

The second wave, the *carotid* or *c-wave,* coincides with the commencement of ventricular systole. It thus marks the commencement of the sphygmic period of the cardiac cycle. The c-wave has been considered a communicated wave from the adjacent carotid artery; but it has also been assumed that the projection of the tricuspid segments into the right auricle during ventricular systole plays a certain rôle in its production.

The third wave, the *ventricular,* or *v-wave,* corresponds to the latter portion of ventricular systole and early diastole. This wave has a variable form and size in different tracings. In its typical form it consists of two elevations, designated v-1 and v-2 respectively, separated by a slight notch. The first elevation occurs during the end of the sphygmic period, whereas the second elevation occurs just subsequent to the completion of this period.

Conflicting views are entertained as to the proper interpretation of the two elements of the v-wave. It is generally accepted that v-1 is due to the accumulation of blood in the veins subsequent to auricular filling, while ventricular systole is still in progress. This view is confirmed by the fact that the magnitude of this wave is increased by rapid auricular filling, whether this be due to augmented venous pressure or tricuspid regurgitation.

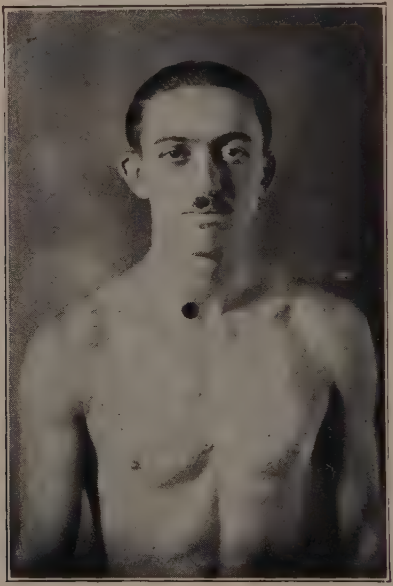


Fig. 116.—Site of jugular bulb. Proper point for recording venous pulse.

V-2 follows the closure of the semilunar valves, and the factors concerned in its generation must operate between the closure of these valves and the opening of the auriculoventricular valves. It has been attributed to the elevation of the auriculoventricular septum during early diastole, and to regurgitation from the auricle into the veins as the heart falls backward from the thoracic wall during this phase of the cardiac cycle.

The notch intervening between the c-wave and the v-wave, termed the *first negative phase,* and commonly designated x, corresponds to auricular diastole. This interval, combined with the duration of the c-wave, represents the period of auricular filling, during which blood is aspirated more rapidly from the veins, resulting in the first negative phase. It is probable that a further factor in the production of this notch is the downward displacement of the auriculoventricular septum during ventricular systole.

The notch immediately following the v-wave, termed the *second negative phase* and commonly designated *y*, corresponds to the opening of the tricuspid valve during early ventricular diastole, leading to more rapid emptying of the cervical veins.

Identification of the Waves.—In the interpretation of polygraphic records it is essential to correctly identify the three basic waves of the phlebogram. This is accomplished by first identifying the c-wave of the tracing. Whether this wave is transmitted from the carotid artery or not, it is certain that it corresponds in point of time with the carotid pulse. As it requires 0.1 second for the pulse wave to be propagated from the carotid to the radial artery, the c-wave is identified by picking out the wave of the phlebogram which occurs 0.1 second prior to the commencement of the anacrotic limb of the radial sphygmogram.

In the identification of the a-wave of the phlebogram it is to be recalled that it requires 0.2 second for the contraction impulse to travel from the auricle to the ventricle. As the appearance of the carotid pulse coincides with the commencement of the sphygmic period, by picking out the wave of the phlebogram which occurs 0.2 second prior to the c-wave, the a-wave of the tracing is identified. In the identification of the waves in this manner it is essential that the several pens of the recording instrument be in accurate alignment or that due allowance be made for any difference in their relative positions. Hence it is necessary to erect upon the tracing two ordinates by lateral movements of the pens to indicate their positions.

Clinical Applications.—Polygraphy may be utilized with advantage in the study of arrhythmia resulting from heart-block, variations in vagus tone, premature contraction of auricle or ventricle, and auricular flutter and fibrillation, as well as in estimating the integrity of the myocardium of the two sides of the heart.

The interval elapsing between the commencement of the a-wave and the c-wave represents graphically the time which is required for the conduction impulse to pass from the auricles to the ventricles; hence, augmentation of this interval points to a lesion of the auriculoventricular bundle. On the contrary, in certain cases of tachycardia this conduction time is observed to be abbreviated.

In right ventricular engorgement the v-wave occurs appreciably earlier in the phlebogram, and in the presence of tricuspid regurgitation it may replace the preceding waves with the production of the positive or ventricular type of venous pulse. In this type of venous pulse, frequently encountered in auricular fibrillation, a-waves and c-waves are absent, as is also the first negative phase. The tracing presents a series of v-waves separated by the second negative phase, *y*.

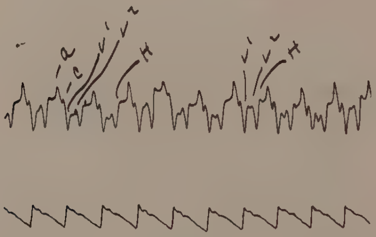


Fig. 117.—Auricular venous pulse with H-wave. Third heart sound upon auscultation.

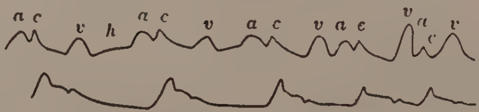


Fig. 118.—Sinus arrhythmia.

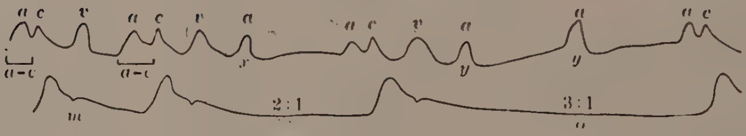


Fig. 119.—Partial heart-block.

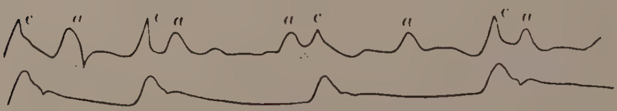


Fig. 120.—Complete heart-block.

Similarly, in right ventricular engorgement there is occasionally noted in the phlebogram a minor wave, the *h-wave,* occurring immediately subsequent to the second negative phase. This wave is attributed to rapid elevation of the cusps of the tricuspid valve, and is produced by the same mechanism which is responsible for protodiastolic gallop rhythm and the third heart sound.

Sinus Arrhythmia.—In sinus arrhythmia, arising as a result of variations in vagus tone, the phlebogram is characterized by a regular sequence of a-c-v-waves, and a normal a-c interval. The disturbance of the rhythm in this instance consists in the early or late occurrence of each group. In individual cases the length of the cycles varies to a striking degree. Thus, in long cycles an H-wave may be introduced, whereas in the shorter ones the v-wave may be combined with the next succeeding a-wave. In extreme cases the a-c interval is apt to vary slightly, but it cannot be definitely stated that this represents an altered conduction time under these circumstances.

Heart-Block.—In the case of incomplete heart-block the earliest stages of the pathologic condition are represented by a lengthening of the a-c interval, representing the time required for conduction of the contraction impulse over the A-V bundle. However, such curves should be corroborated by other evidence of delayed conduction before rendering a positive diagnosis of partial heart-block. Similarly, in the presence of sinoauricular block, impaired conductivity between the S-A node and the ventricle may occur without alteration of the graphic a-c interval. The diagnosis of partial block may be made, however, when an increased a-c interval or a progressively increasing interval is followed occasionally or regularly by a dropped arterial beat. In the accompanying illustration, at the points x and y the a-wave recurs regularly, but is not followed by c-v complexes.

In the presence of *complete heart-block* the jugular tracing exhibits a regular succession of a-waves, interrupted at irregular intervals by c- and v-waves of ventricular origin. These groups fall between the a-waves or are superimposed upon them.

Premature Contractions (Extrasystoles).—In the case of premature contractions of auricular origin the jugular tracing is characterized by an orderly sequence of a-c-v-waves, for an entire a-c-v group occurs prematurely. The early appearance of the premature a-wave causes an interruption of the regular rhythm of the auricle which is initiated by the S-A node. In this rhythm the radial sphygmogram exhibits commonly a premature pulse wave, occurring shortly after the regular pulse wave, and followed by an apparent intermission or pause. Careful measurement of the tracing, however, will reveal the fact that the pause is not fully compensatory; that is, it combined with the premature wave is not equal to the duration of two regular radial pulse waves, a fact which serves to differentiate the rhythm from premature contractions of ventricular origin.

In the presence of premature contractions of ventricular origin the jugular tracing exhibits a premature c-wave, which is followed by an a-wave as the auricle contracts. As this auricular stimulus finds the ventricle in its refractory phase, it is followed by no c-v complex. For this complex is substituted a stasis rise, which fills the compensatory period. The premature contraction may occur so early as to replace the v-wave. Such a contraction is usually so weak, however, that the aortic valve is not opened, and there is a dropping of a wave in the arterial sphygmogram.

The premature contractions which are of sufficient force to open the aortic valve result in a premature radial pulse wave, which is followed by a pause or apparent intermission of the pulse. In this event the subsequent pause is fully compensatory; that is, the pause combined with the premature contraction is exactly equal to two regular pulse waves.

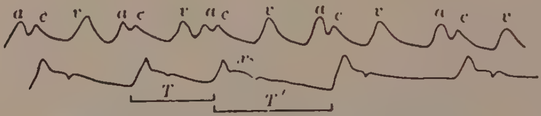


Fig. 121.—Premature auricular contraction.

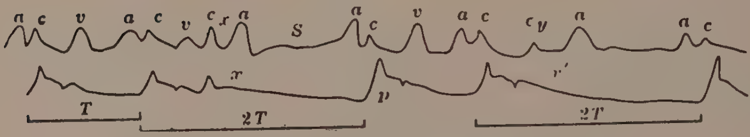


Fig. 122.—Premature ventricular contraction.

Paroxysmal Tachycardia.—In the presence of paroxysmal tachycardia the polygraphic tracings exhibit a regular sequence of a-c-v-waves in the jugular pulse, with a very much abbreviated a-c interval. Not infrequently one or more premature auricular contractions are in evidence at the inception and subsidence of the attack. Occasionally in cases of extreme rapidity, the c-wave becomes merged with the succeeding v-wave.

Auricular Flutter.—In cases of auricular flutter in which the ventricular rate is not unduly accelerated, the jugular tracing frequently exhibits a rapid succession of small a-waves, which are occasionally interrupted by waves of ventricular origin. When the ventricle beats rapidly, however, and tricuspid regurgitation occurs, all evidence of auricular contraction is obscured, and the ventricular form of venous pulse alone is in evidence. In such cases a diagnosis is not to be based upon polygraphic tracings.

Auricular Fibrillation.—In the presence of fibrillation of the auricle the jugular tracing is characterized by an entire absence of a-waves and the presence of ventricular c-v complexes. These waves have a variable conformation and they recur very irregularly, but at a rapid rate. Between these systolic groups there are to be noted lengthy stasis periods, upon which small undulations may be evident. Other indefinite waves are noted which probably are caused by ineffective contractions of the ventricles.

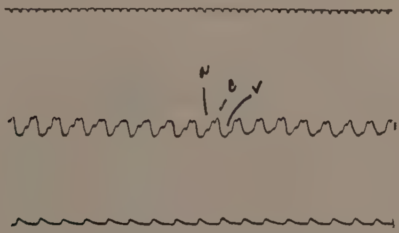


Fig. 123.—Paroxysmal tachycardia.

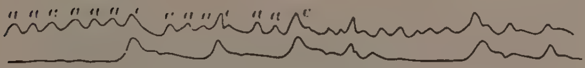


Fig. 124.—Auricular flutter.

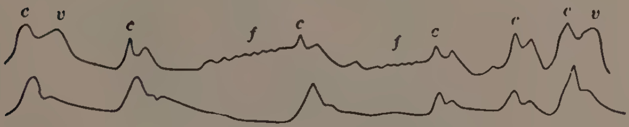


Fig. 125.—Auricular fibrillation.

The radial sphygmogram exhibits a totally irregular rhythm with intermissions, which correspond to the pulse deficit which is to be demonstrated upon auscultation of the precordia.

For detailed descriptions of the polygraphic findings in cardiovascular disease, the reader is referred to the works of MacKenzie, Lewis, Hay, Lamson, and Wiggers, upon the graphic registration of the heart beat.

The electrocardiogram has replaced the polygram to a considerable extent for the study of the arrhythmias.

SPHYGMOMANOMETRY

Blood pressure may be estimated by several types of sphygmomanometer, the procedure constituting sphygmomanometry. For practical work one of the modifications of the Riva-Rocci instrument will be found generally satisfactory, the basis of all sphygmomanometers of this type consisting of an inflatable rubber bag contained in an inelastic cuff, a mercury manometer, and an air pump so connected by rubber tubing that the air which is pumped is distributed with equal pressure to the cuff and the manometer. A further type of instrument, which dispenses with the use of the mercury manometer, and which instead of recording the pressure in millimeters of mercury records the pressure upon a dial, is the Rogers Tycos sphygmomanometer.

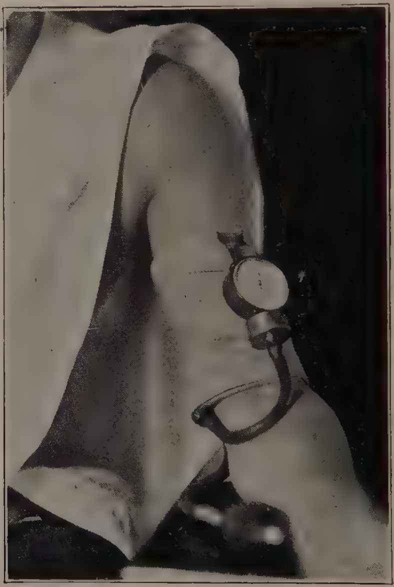


Fig. 126.—Rogers Tycos dial sphygmomanometer.

With either type of instrument, in recording the blood pressure two methods may be employed; namely, the palpatory method, and the auscultatory method. Whichever method is employed, certain details of the technic must be observed in order to obtain accurate results. The pressure is usually recorded from the brachial artery during the routine examination, as it is readily accessible and is almost on a level with the heart. The cuff should be applied to the arm at least two inches above the bend of the elbow; the connections of the tubing to the different portions of the instrument should be air-tight; the dilatable rubber bag should be carefully adapted to the inner aspect of the arm, overlying the brachial artery; and the cuff should be snugly applied, but not with sufficient force to interfere with the venous return. No fabric should be permitted to intervene between the cuff and the integument; and the clothing of the extremity should be removed in order to avoid constriction of the artery by rolling up a sleeve.

Clinically it is desirable to estimate the blood pressure during ventricular systole (systolic pressure); during ventricular diastole (diastolic pressure); and to determine the difference between these figures (pulse pressure).

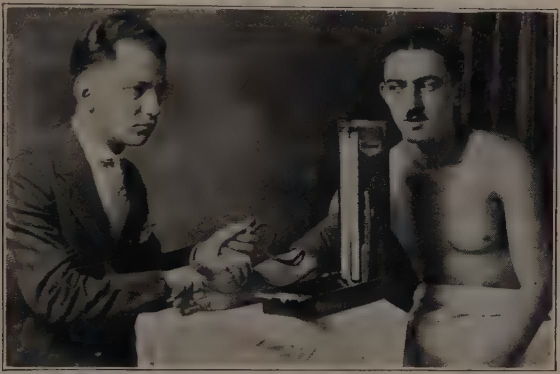


Fig. 127.—Estimation of blood pressure by palpatory method.

Palpatory Method.—With the instrument properly adjusted to the arm, air is slowly pumped into the cuff and manometer until the pulse becomes inappreciable to the fingers palpating the radial artery. When the pulse disappears, the mercury is pumped up 10 or 15 millimeters above this point; and the release valve is turned, allowing the mercury column to slowly descend. At the instant that the pulse becomes again appreciable at the wrist the release valve is closed, and the *systolic pressure* is read upon the scale or dial.

The principle involved in this procedure is that it requires an amount of external pressure to obliterate the pulse in the artery which is commensurate with the intravascular pressure during systole. It is doubtless true that a certain degree of the external pressure is consumed in overcoming the resistance of the soft tissues, the arterial wall and the blood velocity; but in the normal adult subject this difference is estimated at 7 to 10 millimeters of mercury. By the use of a broad cuff, errors in this direction are reduced to the minimum.

Having determined the systolic pressure by the method described, the release valve is again slowly rotated and the mercury column allowed to descend upon the scale, the undulations of the column being closely observed. The descent is attended by rhythmic oscillations; and at one point in the descent these oscillations become very pronounced, this point corresponding to the *diastolic pressure,* and as a rule being accompanied by a larger pulse wave than normal at the wrist.

By deducting the diastolic pressure from the systolic pressure, the *pulse pressure* is obtained.

Auscultatory Method.—The auscultatory method of estimating the blood pressure is more accurate than is the palpatory method; and, in addition, it shows wider ranges of pressure in the individual case. The systolic pressure, as determined by the auscultatory method, is always from 7 to 10 millimeters above that registered by the palpatory method, whereas the diastolic readings range from 10 to 15 millimeters below those obtained by the palpatory method.

In estimating blood pressure by this method the bell of the stethoscope is applied over the brachial artery just above the bend of the elbow, and the cuff is inflated until all sound disappears. Having attained this point, the air is allowed to slowly escape from the cuff, whereupon a series of sounds are audible, which have been divided into five phases.

The *first phase* is represented by the first sound which is audible, and represents the proper point at which to record the *systolic pressure.* The sound is attributed by Erlanger to the water-hammer action of the blood column at the instant of its contact with the relatively stationary column below the point of constriction. The first phase is quickly followed by a peculiar murmuring sound as the tension in the cuff is lowered, the *second phase,* which is attributed to the induction of "fluid veins" in the blood stream as it passes the point of constriction to enter the wider lumen of the artery beyond the cuff. The second phase in turn is followed by an accentuation of the murmur, constituting the *third phase,* in which a vibration of the vessel wall is assumed to be added to the fluid veins of the second phase. The sharp murmur of the third phase soon gives place to a less intense sound, the *fourth phase,* as the compressed artery gradually returns to its normal cylindrical contour. The fourth phase lasts until all sound ceases, the *fifth phase.*

The *diastolic pressure* may be recorded at the beginning of the fourth phase or at the beginning of the fifth phase, the time at which all sound ceases. There is a difference of approximately 6 millimeters, as the record is made at the fourth or the fifth phase; but as it is often difficult to say just when the fourth phase begins, and as it is relatively easy to determine when all sound ceases, it is a safe rule to record the diastolic pressure at the fifth phase, bearing in mind the discrepancy between the reading at the two phases.

Goodman and Howell place the duration of the first phase at 14 millimeters, the second at 20 millimeters, the third at 5 millimeters, and the fourth at 6 millimeters. They hold that prolongation of the third phase indicates a powerful ventricular systole and arterial fibrosis, and that as the pressures approximate one another in a failing heart the second phase is the earliest to show abbreviation.

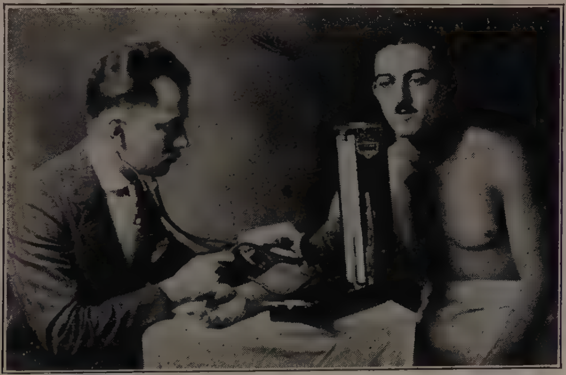


Fig. 128.—Estimation of blood pressure by auscultatory method.

The Sphygmotonograph.—An instrument has recently been devised for automatically determining blood pressure, the results being recorded by a writing pen upon a circular paper chart. This apparatus is known as the sphygmotonograph.

The chart movement is actuated by a metallic diaphragm which is governed by falling air pressure. The speed is regulated by an automatic air leak. No clock or motor is employed. The tracings are inscribed directly upon a pressure scale calibrated in millimeters of mercury.

Method of Use.—An arm bag which has two separate chambers is carefully wrapped on the patient's arm above the elbow. The upper chamber leads through an air tank to the front side of the recording mechanism, and the lower chamber leads directly to the opposite side.

As a result of this, the pen moves in an inward direction in response to each impulse received from the upper chamber, but reverses itself, moving outward when the impulse originates in the lower chamber.

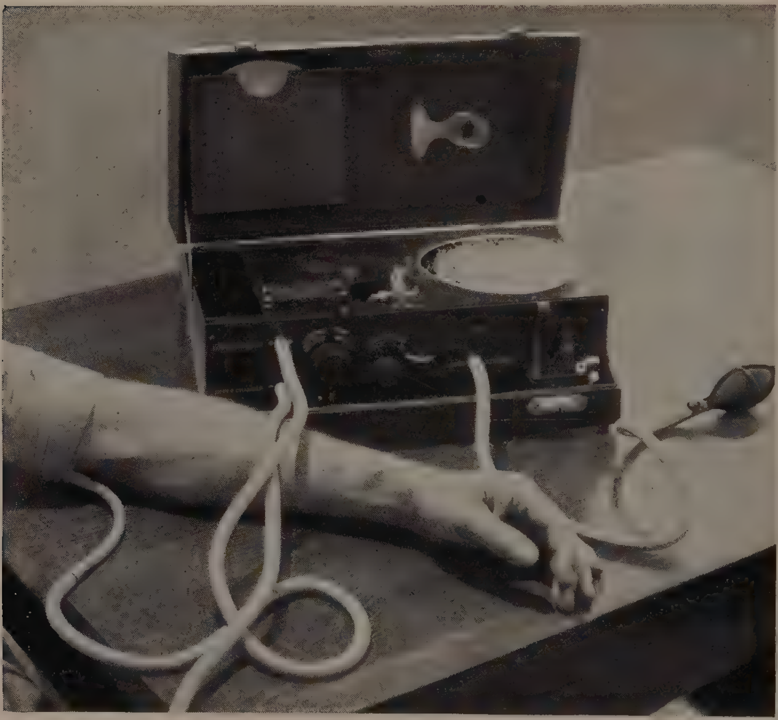


Fig. 129.—The recording blood pressure machine.

When the instrument is inflated, the recording mechanism is protected from inrushing air by the closure of a hand valve.

After inflation has been carried to a point sufficiently high to obliterate the artery, the recording mechanism is connected with the remainder of the system, and the inked pen, which is attached directly to the recording diaphragm by a magnifying linkage, is lowered to a writing position on the chart.

An automatic air leak is opened, which provides an increasing larger orifice as the pressure in the whole system decreases. This tends to maintain uniformity of the chart's rotation.

The speed of rotation is governed by an adjustable dial which is set in accordance with the patient's pulse rate, thereby making all readings easily comparable.

The chart slowly rotates, pausing momentarily with each pulse beat, when the automatic air leak is opened. This makes the record appear as a series of spikes.

The pen inscribes short spikes toward the center of the chart in response to each pulse beat, prior to the systolic point. Their size is due to the cushioning effect of the air tank through which they must pass.

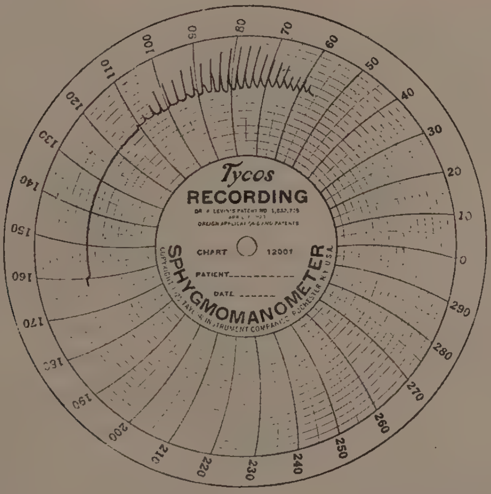


Fig. 130.—Normal blood pressure tracing. Systolic pressure 111; diastolic pressure 70.

The pulse wave, upon arrival at the point of systolic pressure, passes for the first time underneath the upper chamber and strikes the lower section of the arm bag. The impulse set up is transmitted directly to the rear side of the recording mechanism. This causes the pen for the first time to move in an outward direction upon the chart. The pen continues writing in an outward direction, inscribing a spike for each pulse wave passing underneath the lower chamber of the arm bag.

It must be remembered that each spike is made under different conditions. The decreasing pressure in the arm bag causes the spikes to gradually increase in length until the maximum movement has been reached, following which a diminution in length occurs which indicates the diastolic zone.

The Sphygmotonogram.—The sphygmotonogram, as made with the Tycos recording sphygmomanometer, is the curve traced by the arterial pulse under a gradually decreasing pressure from obliteration to normal extra-arterial compression.

This curve exhibits characteristics common to both the polygraph and electrocardiograph, but in addition contains certain new features peculiar to itself alone. Chief among these is the ability to accurately identify the exact blood pressure in graphic form.

Systolic pressure is identified at the point in the tracing where the pen reverses the direction in which it is writing, moving for the first time toward the outside of the chart.

The diastolic pressure is established at the point directly following the maximum excursion of the pen where several spikes tend to become of nearly equal length.

The sphygmotonogram also identifies cardiac irregularities affecting the pulse, their diagnosis resting largely upon the relation of one beat to another, rather than on the study of the single pulse wave.

The amplitude of the recorded beat, with uniform application of the compression sleeve, depends entirely upon the pulse characteristics of the patient. The length of the pen's movements, therefore, gives information of diagnostic significance.

Normal Pressures.—It is difficult to establish definite figures which represent normal blood pressures at different ages, in the two sexes, and in various walks of life. In a general way, normal pressures bear a fairly definite relation to the age of the subject, but the pressures are modified by coincident changes in the circulatory system, which have no definite relation to the age of the patient. It has been stated that the normal systolic pressure for a person twenty years of age is approximately the age plus one hundred, or 120 millimeters, and that for each two succeeding years of age the normal pressure increases one millimeter. Sex, weight, and habitus cause such marked variations that a rule such as the above is of little value and often causes a patient considerable anxiety should he vary from this arbitrary standard.

The majority of cardiologists regard systolic pressures of 120 to 130 millimeters and diastolic pressures of 80 to 90 millimeters as within the normal range in patients up to forty years of age.

Diastolic pressures above 90 are regarded with suspicion, and a diastolic measurement of 100 millimeters is distinctly pathologic. Pulse pressures usually vary between 30 and 50 millimeters in normal subjects, and readings above 50 are viewed with suspicion. Very large pulse pressures usually point to aortic regurgitation or chronic nephritis.

Normal Variations.—Before drawing conclusions from variations in blood pressure, certain normal variations must be eliminated. Thus, the pressure varies with the attitude assumed by the patient, being higher when he stands, and lower when the sitting or recumbent attitude is assumed. So constant is this variation in the normal subject that its absence is adequate proof of vasomotor instability. Clinically the blood pressure may be estimated with the subject in the sitting or recumbent posture with equally satisfactory results; but whichever attitude is assumed at the first examination should be employed in all subsequent estimations.

After the ingestion of a full meal the blood pressure is slightly higher than it is several hours after a meal; and during sleep it is normally lower than during waking hours. Exercise, nervous excitement, and the ingestion of stimulants, all increase the blood pressure temporarily. During childhood it has been shown that blood pressure is proportionate to the body weight rather than to the age of the subject; but with advancing age in the adult subject there is a progressive increase in blood pressure.

Pathologic Variations.—When a high *systolic pressure* is encountered in a patient who is and has been in repose, it points to cardiac hypertrophy, the causes of which are to be sought. Chronic nephritis, whether a glomerulonephritis or a diffuse sclerosis of the kidney; arteriosclerosis, or increased intracranial pressure are common causes. In the pregnant woman it may point to threatened eclampsia. In cardiovascular disease a high systolic pressure which is accompanied by an increased pulse pressure usually is indicative of adequate compensation; whereas a normal or lowered systolic pressure with a diminished pulse pressure points to threatened cardiac failure.

In the presence of autointoxication the systolic pressure is frequently elevated as the result of the elaboration of toxic bases in the gastrointestinal tract.

A decrease in the systolic pressure accompanies extensive loss of blood, internal or external, malnutrition, the cachexia of malignant disease, chronic alcoholism, myocardial degeneration, pericardial effusion, orthostatic albuminuria, and the vascular asthenia of Addison's disease.

The importance of variations in the *diastolic pressure* has come to be generally recognized. The diastolic pressure represents the degree of peripheral resistance which must be overcome before the left ventricle may discharge its contents; and if this pressure is raised, as it is in arterial fibrosis and chronic renal inflammation, an added burden is thrown upon the heart before the blood can begin to circulate. Moreover, when a high diastolic pressure is encountered, it is of the first importance from the standpoint of prognosis and treatment to determine whether it is accompanied by an increase in the systolic pressure, or whether the increase is due to functional angiospasm. In aortic regurgitation the diastolic pressure is low, while the systolic pressure is commonly increased, giving a very large pulse pressure. In toxic goiter the diastolic pressure is likewise lowered, with an approximately normal systolic pressure.

The *pulse pressure* deserves careful study in all cases in which sphygmomanometry is practiced. The pulse pressure represents the contractile power of the left ventricle in excess of the diastolic pressure; or, in other words, the power of the left ventricle over and above the peripheral resistance. During the course of lobar pneumonia, when the heart is laboring under an increased load, a daily record of the pulse pressure gives valuable information as to the state of the myocardium, and affords a valuable prognostic and therapeutic index. As the overtaxed heart fails, the systolic pressure gradually approximates the diastolic pressure, the pulse pressure steadily diminishing until the point is reached at which the coronary circulation is inhibited and the nuclear centers are no longer supplied with blood, the point at which life becomes impossible. A systolic pressure of 60 millimeters seems to be the minimum pressure compatible with life, although lower pressures are recorded in the earlier literature.

Venous Pressure.—Hooker and Eyster have devised an apparatus for the estimation of the venous pressure in the veins of the forearm. The instrument consists of a box with aluminum frame with a glass aperture upon the upper side, while the lower side is composed of rubber dam, which is in contact with the forearm. The instrument is connected with a water manometer and air pump and the venous pressure is recorded in centimeters of water. Hooker and Eyster find a progressive rise in venous pressure from youth until old age, and that impairment of the myocardium, attended by lowering of the systolic pressure is attended by a concomitant rise of venous pressure.

Villaret finds that normal venous pressures are represented by 12 centimeters of water for females, and by 14 centimeters for male subjects; and Clark holds that a pressure of 20 centimeters is of grave prognostic significance, representing the limit between compensation and decompensation of the heart.

Clinically, an elevation of venous pressure is noted in three conditions: (1) in aortic regurgitation, (2) in arterial hypertension due to chronic nephritis, and (3) in cases of cardiac insufficiency from myocardial degeneration.

THE FOREARMS AND ARMS

The muscular development of the forearms should be compared and any variation of muscular activity noted when the hands are opened and closed.

With the hand cupped over the elbow joints, the subject is told to flex the forearms, thus any restriction of joint movements or roughness may be determined and a comparison made with the joints of the opposite side. After fractures at the elbow, restriction of motion is common, sometimes accompanied by a catch or lock of the joint due to loose cartilage or fragments of bone. X-ray examination is often necessary to establish a diagnosis of diseased or injured bones or joints.

The notch above the inner condyle of the humerus contains the epitrochlear lymph glands. These glands are frequently enlarged from syphilis and occasionally from tuberculosis. Infections of the hand and forearm rarely cause enlargement of the epitrochlear glands.

Upper arm development and muscle movements are compared as in the case of the forearms. Ruptured biceps produce two tumor masses with a sulcus between when an attempt is made to contract the muscle.

Palpation at the point of insertion of the deltoid muscle will elicit pain in many normal individuals. This pain is quite severe in subdeltoid bursitis.

The brachial artery is palpable along the inner surface of the arm, usually about the middle or third. In the aged this artery is usually thickened and tortuous. When the aortic valve is incompetent it is often felt to jerk with each systole of the chest.

The axilla is palpated by pushing the fingers firmly into the axillary summit and rolling the subcutaneous tissues under the fingers. Enlarged glands in the axilla may result from pyogenic infections of the hands and arms or axillary hair follicles, metastasis from newgrowths in the breast, Hodgkin's disease, lymphatic leucemia, and tuberculosis.

Enlarged glands from pyogenic infections and those caused from tuberculosis often break down, forming large fluctuating tumors or discharging abscesses.

The glands in leucemia usually are discrete, but may reach considerable size. In Hodgkin's disease the glands are frequently matted together, forming large irregular tumors.

The shoulder movements are examined as are those of the elbow by cupping the hand over the shoulder and instructing the subject to move the arm in all directions. Arthritis, old dislocations or fractures often restrict joint movement, while roughness or grating can be felt in chronic arthritis or periarthritis. Comparison of the shoulders is, of course, essential, and x-ray examination may finally be necessary before a diagnosis is established.

CHAPTER XVIII

PALPATION OF THE THORAX

Object and Technic.—Palpation is employed in physical examination of the thorax to confirm the findings upon inspection as to the size and shape of the thorax, the respiratory movements and degree of expansion of the chest, and to detect slight deficiencies of expansion which are so slight as to have escaped detection during inspection. Palpation is also employed in the elicitation of several types of vibration or *fremitus* arising in the thorax during health and in diseased states. The systematic and skillful practice of palpation also reveals the degree of resistance of certain lesions developing within the thorax, as also the presence of local tenderness, local areas of pulsation, and vibration in the cardiovascular mechanism. Moreover, palpation yields information as to the condition of the skin and subcutaneous tissues of the thorax, as well as of the ribs and intercostal spaces.

As employed in the routine physical examination, palpation consists in the application in the first instance of the palmar surfaces of the hands to the surface of the thorax for the purpose of appreciating and analyzing variations in the tactile impressions which are conveyed to the palpating hands. This maneuver is employed in the examination of the anterior, lateral, and posterior regions of the thorax. In palpation of the supraclavicular and infraclavicular regions, on the contrary, as well as in palpation of the intercostal spaces, palpation with the finger tips alone is more serviceable.

Certain clinicians recommend the employment of the ulnar border of the hand in preference to the palm in the practice of palpation (linear palpation); but, as a rule, the tactile sensations which are appreciated by this portion of the hand are not as fine as are those which may be detected by the more sensitive palms. Again, certain clinicians have adopted palpation with the dorsal aspect of the fingers, holding that the dorsal surface is more sensitive than is the palmar surface of the hand.

During palpation of the thorax the clothing should be removed to the waist so that no fabric may intervene between the hands of the examiner and the surface of the thorax of the subject. The room should be comfortably heated in order to avoid fibril-

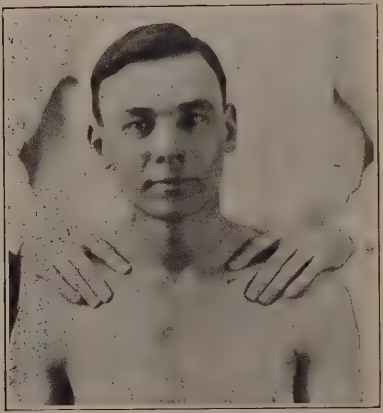


Fig. 131.—Palmar palpation of the apices.

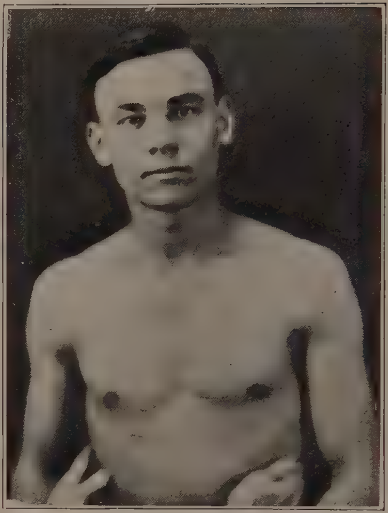


Fig 132.—Palmar palpation of the bases.

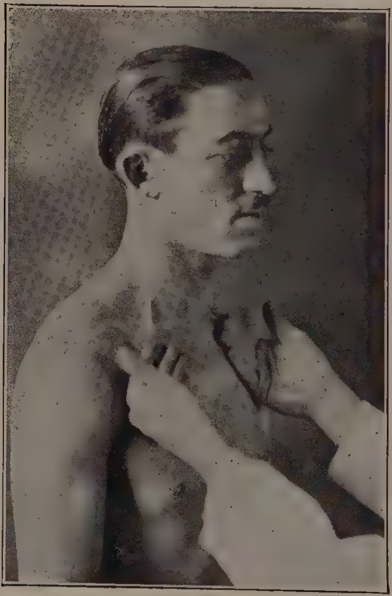


Fig. 133.—Dorsal palpation of thorax.

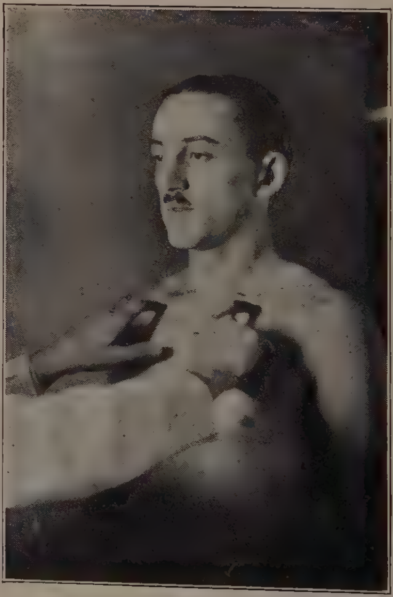


Fig. 134.—Finger-tip palpation of the apices.

lary contraction of the thoracic muscles due to chilling of the surface of the body; and the position of the patient should be natural and free from muscular tension. The hands of the examiner should be warm, and they should be applied firmly and evenly to the area under examination. Palpation of the thorax may be practiced with the patient in the sitting or recumbent posture, preferably in the former. During examinations in the recumbent posture, it is essential that the patient be turned a sufficient number of times to insure palpation of all portions of the thorax.

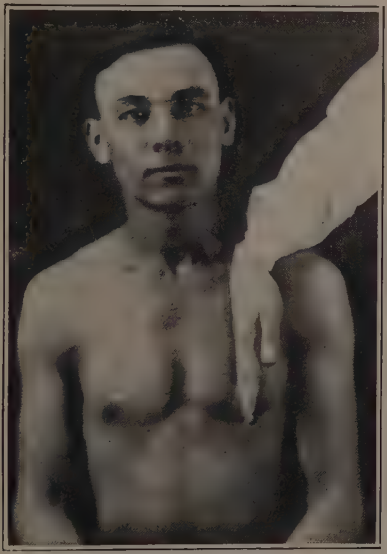


Fig. 135.—Linear palpation of thorax.

THORACIC EXPANSION

In testing the expansion of the pulmonary apices the examiner should stand behind the patient, and with the index and middle fingers in the supraclavicular and infraclavicular regions respectively, he should note the position of these regions during inspiration and expiration. During this maneuver the finger tips should rest very lightly upon the integument, as strong pressure frequently suffices to obscure slight lagging at one apex.

The degree of expansion, as elicited in this manner, is observed to vary widely in different pathologic states. In the presence of an early active tuberculous lesion involving one apex, it may be noted that expansion upon the affected side of the thorax is perceptibly delayed and that it never equals that of the sound side at the completion of inspiration. In this event the deficiency of expansion may be attributable to spasm of the muscles of the shoulder girdle in the absence of extensive destruction of pulmonary tissue. In the case of extensive consolidation of an upper lobe, associated with pleural adhesions or pneumothorax, the affected side of the thorax may remain immobile during inspiration. In such event characteristic changes will be noted in the thoracic integument and musculature, as will be observed in a subsequent paragraph.

In testing for deficient expansion of the infraclavicular and upper mammary regions of the thorax, the examiner should stand in front of the patient; and, placing the finger-tips lightly upon the thoracic integument, observe the relative position of the fingers of the two hands during inspiration and expiration.

In the detection of unequal expansion of the lateral regions of the thorax, the examiner should assume a position in front of the patient; and, applying the finger-tips to the lower lateral regions of the thorax, note their movements during inspiration and expiration. Similarly, in testing the anteroposterior expansion, he should stand beside the patient; and, placing one hand upon the middle of the sternum and the other between the scapulæ, note the degree of expansion at the completion of inspiration.

Palpation, to be serviceable, must be systematic, the entire surface of the thorax being gone over in orderly succession and corresponding regions upon the two sides should be compared. Hasty and unsystematic palpation is a frequent source of erroneous conclusions.

THORACIC VIBRATIONS

Palpable vibration of the thoracic wall (fremitus) is produced by the vibrations of the vocal cords during phonation, by the movement of the roughened surfaces of an inflamed pleura, by the action of coughing, by the forcible movement of fluid in the pleural cavity, and during the percussion of the thorax in the presence of hydatid cyst. These various forms of tactile fremitus are manifested in characteristic manner, and each form furnishes valuable data upon which the examiner may base conclusions.

VOCAL FREMITUS

Vocal fremitus is manifested in the form of a palpable vibration of the thoracic wall, which is conveyed to the palms when

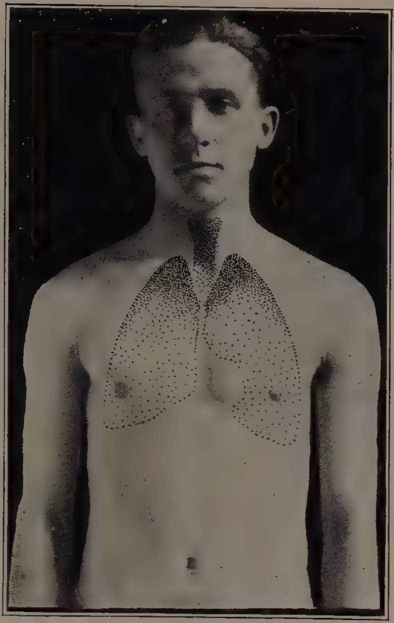


Fig. 136.—Normal variations in vocal fremitus.

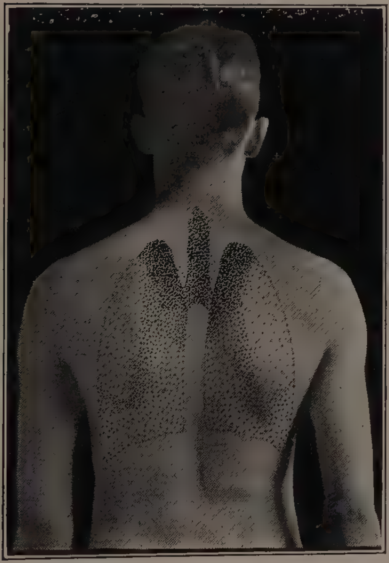


Fig. 137.—Normal variations in vocal fremitus.

these are applied to the surface of the thorax during phonation. The vibration, which has been likened to the sensation which one experiences upon placing the hand upon a resonating box in which a tightly stretched wire or cord is caused to vibrate rapidly, is appreciated immediately upon the act of phonation. Its duration corresponds almost, but not quite, exactly to the duration of the spoken words, the tactile fremitus being prolonged during a very brief space of time after the cessation of speech on the part of the subject.

These vibrations, which originate in the vocal cords of the larynx, and which are the physical basis of the production of the voice, are communicated directly to the two columns of air situated respectively above and below these cords. The aerial column situated below the true vocal cords constitutes a continuous conducting medium throughout the larynx, trachea, bronchi, bronchioles, and pulmonary alveoli, a medium which thus extends to the periphery of the lungs. From the pulmonary alveoli the vocal vibrations are conducted by way of the alveolar walls and the investing pleura to the thoracic walls, and by these walls to the palpating hand placed upon the surface of the thorax.

As the air passages form a closed system, the vibrations arising in the vocal cords are naturally conducted downward, since the lateral propagation of the waves is effectually prevented by the walls of the tubes. Doubtless the solid walls of the air passages are also concerned to a minor degree in the conduction of the vocal vibrations along their course to the thoracic wall; but in this function these structures can play only a minor rôle, in accordance with the physical principle that the transmission of vibrations along a solid structure is enfeebled in direct proportion to the degree of variation in the structure of the conducting medium. Certainly the waves of vibration encounter numerous adverse changes of structure in the course of their conduction by way of the solid structures from their origin in the larynx to the termination of the finer bronchioles in the periphery of the lung.

In eliciting vocal fremitus the examiner should apply the palms or the ulnar borders of the hands to the surface of the thorax while the patient is directed to count ''one, two, three,'' or to repeat the words ''ninety-nine'' in a deep voice of uniform intensity, the examiner meanwhile noting the intensity with which the vibrations are transmitted to the palpating hand in the various regions of the thorax.

The intensity of vocal fremitus in the normal subject is governed by the intensity of the voice during phonation; by the pitch of the speaking voice; by the variations in the relations of the primary bronchi to the thoracic wall; and by the varying thickness of the thoracic wall in different regions of the thorax.

The intensity of vocal fremitus varies directly with the intensity of the speaking voice. Just as the voice is stronger in proportion as the amplitude of each vibration is greater, so also are vocal vibrations of greater amplitude manifested by tactile vibrations of corresponding intensity. If, during palpation of the thorax, the patient is directed to speak continuously in a voice of progressively increasing intensity, the resulting vocal fremitus will be observed to become progressively more intense. If, on the contrary, the speaking voice is so modulated as to become progressively less intense, there is witnessed a corresponding diminution in the degree of tactile fremitus, which indeed is completely abolished when the voice is reduced to a whisper. Hence, it is of the first importance during the elicitation of vocal fremitus for diagnostic purposes to see that the subject speaks in a voice of uniform intensity throughout the examination.

The pitch of the voice exerts a striking influence upon the intensity of vocal fremitus, the tactile vibration being most intense in subjects with deep, bass voices, and possessing the minimum of intensity in subjects of high suprano voices. Indeed if, in a suitable subject, the examiner will palpate the thorax during phonation of progressively ascending pitch, in the case of the high suprano voice, a point will be reached at which the tactile fremitus disappears. The underlying physical basis of this progressive enfeeblement of vocal fremitus resides in the fact that as the pitch of the voice ascends, the sound waves become progressively more rapid with a corresponding diminution in their amplitude, until the point is reached at which palpable vibration of the thoracic wall is no longer produced. Upon these physical principles it is readily understood that vocal fremitus is normally more intense in the adult male subject with his naturally deep voice called forth by waves of moderate rate and wide amplitude; and that it is correspondingly less intense in the adult female subject and in children, in whom the voice possesses less volume but gains in pitch, or, in other words, in whom the sound waves possess a more limited amplitude but a higher rate of vibration.

Vocal fremitus in the normal subject is slightly more intense upon the right side of the thorax than upon the opposite side, because of the greater caliber of the right bronchus and because of its anatomical situation in closer proximity to the anterior thoracic wall than that of the left bronchus. It follows that during phonation a more considerable column of air is set in motion in the right bronchus, and its closer relation to the thoracic wall further facilitates the transmission of the vibrations to the palpating hand. Vocal fremitus is naturally most pronounced in those regions of the thorax where the larger bronchi approach the thoracic parietes; and it exhibits impairment in those areas where the bronchi are separated from the chest wall by the intervention of aerated pulmonary tissue. Hence, it presents its maximum intensity in the root of the neck, immediately over the course of the larynx and the trachea, and is more intense over the right apex than upon the corresponding area of the opposite side.

The condition of the thoracic wall exercises a considerable influence upon the intensity of vocal fremitus. The deep, muscular thorax of the robust subject and the thorax which is clothed with a thick paniculus adiposus in the obese subject present a striking enfeeblement of the vocal vibrations; whereas these are unduly intense upon palpation of the thin, poorly muscled thorax of the emaciated subject, as well as in the thin and elastic thorax of the female subject and the child. Extensive edema and local suppuration of the thoracic wall similarly mask the integrity of the tactile vibrations. Upon more minute examination, it can usually be demonstrated that vocal fremitus is normally more intense over the intercostal spaces than it is in the regions which overlie the ribs. It is very natural that the intercostal musculature should respond more readily to the intrathoracic vibrations than the less elastic osseous ribs.

Vocal fremitus presents certain *regional variations* in the intensity with which it is transmitted to the palpating hand in various regions of the thorax. Considering the thorax as a whole, the fremitus in the normal subject is most intense upon the anterior thoracic wall; it is slightly less intense upon the lateral walls; and it exhibits its minimum of intensity upon the posterior wall of the thorax. A number of variations in the relative intensity of the fremitus in the normal subject are to be encountered upon each of these three aspects of the thorax.

Upon the anterior surface of the thorax the fremitus is less intense in the supraclavicular region, which corresponds to the anterior surface of the apex of the lung, than upon any other portion of this surface with the exception of the narrow zone immediately overlying the bony clavicle and over the distribution of the mammary gland in the female subject. In the supraclavicular region the fremitus becomes progressively more intense as the examiner palpates inward toward the median line, owing to the proximity of the trachea to the inner portion of this region.

Upon palpation of the narrow zone immediately superjacent to the clavicle, the tactile fremitus is greatly enfeebled, contrasting in a striking manner with the more pronounced vibrations which are encountered in the supra- and infraclavicular regions above and below the bone. But even this limited area of the thoracic surface presents certain variations in the intensity of the fremitus, which exhibits its greatest strength upon palpation of the inner third of the clavicle, near its junction with the sternum. In the middle third of the bone the fremitus exhibits a moderate diminution over that of the inner third, a diminution which progressively increases in the outer third of the bone as the palpating hand approaches the scapula.

Upon passing from the clavicular area to the infra-clavicular region, there is a very appreciable reinforcement of the fremitus. This intensity is moderately reduced in the mammary region in the male subject, and quite markedly reduced over the mammary gland in the female subject.

In the areas in which the heart and the liver come into direct contact with the anterior thoracic wall there is an abrupt cessation of vocal fremitus. Linear palpation with the ulnar border of the hand is very serviceable in accurately determining the precise points at which the fremitus terminates in relation to these organs.

In the median line of the thorax, in the sternal region, at least three variations in the intensity of vocal fremitus may be detected. In this region the fremitus is most feeble over the upper portion, which corresponds anatomically to the manubrium sterni; it attains its maximum strength over the gladiolus; and it is again enfeebled in the lower portion of the region, which overlies the ensiform cartilage.

In the ventral aspect of the neck, in the region in the median line which overlies the larynx and trachea, vocal fremitus is elicited in its purest form. Even in this restricted region, however, certain variations in its intensity may be appreciated by careful palpation. Its point of maximum intensity corresponds to the level of the inferior border of the thyroid cartilage, from which point the vibrations become progressively enfeebled as the palpating hand travels upward toward the floor of the mouth and downward toward the episternal notch.

Upon the lateral thoracic walls vocal fremitus is most intense in the upper portion of the axillary region, becoming progressively more feeble as one palpates downward over the lower axillary and infraaxillary regions. Upon the right side of the thorax the fremitus is interrupted abruptly at the upper border of the eighth rib by the apposition of the liver with the thoracic wall. Similarly, upon the left lateral aspect of the thorax the fremitus is interrupted at the upper border of the ninth rib by the apposition of the spleen with the thoracic wall.

Upon the posterior wall of the thorax vocal fremitus presents its greatest intensity in the interscapular regions, overlying the trachea and its bifurcation to form the two primary bronchi. In the subscapular regions, overlying the pulmonary bases, the fremitus undergoes a considerable diminution in intensity by reason of the considerable distance of these regions from the site of production of the vocal vibrations. In the regions overlying the scapulæ the fremitus is more feeble than elsewhere upon the posterior thoracic wall, the point of minimum intensity corresponding accurately to the course of the spine of the scapula.

Pathologic Variations

In the presence of disease of the respiratory organs numerous gradations in the intensity of vocal fremitus are encountered. As a result of morbid changes in the bronchi, lungs, pleura, or thoracic wall, the fremitus may be found to be increased in intensity, to be diminished in intensity, or to be entirely abolished over certain regions of the thorax. In general, affections of the pleura are attended by diminution of vocal fremitus, while disease of the lungs is manifested by exaggeration of the fremitus; but in the interpretation of the findings in any case it is to be remembered that affections of the bronchi and of the thoracic wall are capable of causing either diminution or reinforcement of vocal fremitus independently of disease of the lung or the pleura.

Increased Vocal Fremitus.—Vocal fremitus is increased in intensity in all conditions which are attended by solidification of the pulmonary tissues with a concomitant diminution in the air content of the area in question. This condition of solidification and anaeration obtains in the consolidations of pneumonia, phthisis, and hemorrhagic infarction of the lung; in the presence of diffuse carcinomatous infiltration of the lung, provided that this infiltration does not cause bronchial occlusion; in pulmonary atelectasis from external pressure; and in indurative contraction of the lung from fibroid phthisis, chronic interstitial pneumonia, or pulmonary syphilis. It is essential, however, for consolidations to yield exaggerated tactile fremitus, that the consolidated areas be situated in the periphery of the lung, and that the bronchi which terminate in the consolidated areas be free from occlusion.

The physical basis for the exaggeration of vocal fremitus under these circumstances is readily appreciated, since a continuous medium of uniform structure conducts vibrations with far greater intensity than does a conducting medium which is constantly undergoing variations in structure, as are the normal pulmonary alveoli, in which thin walls of pulmonary tissue constantly are interrupted by minute air-containing spaces. When a solid neoplasm of the lung occupies the peripheral portion of this organ and at the same time extends so deeply into the lung as to come into contact with one or more of the larger bronchi, the physical conditions are eminently favorable for the transmission of vocal fremitus to the surface of the thorax with the maximum degree of exaggeration.

In the presence of compression of the pulmonary tissues by a pleural effusion, the exaggeration of vocal fremitus is ordinarily masked over the lower portion of the thorax, corresponding to the area occupied by the effusion, only to be increased in intensity in the region immediately above the level of the effusion, as a result of compression and relaxation of the pulmonary tissues in the upper portion of the pleural cavity. It is quite otherwise in the case of pulmonary compression by an extensive pericardial effusion. In this instance the compression operates upon the internal surface of the inferior lobe of the left lung, leading to exaggeration of vocal fremitus over the compressed lobe. Excessive abdominal distention in ascites, meteorism, or large abdominal tumor, can operate in a similar manner, and exhibit exaggeration of vocal fremitus over the lower segment of the thorax.

Pulmonary cavities with free communication with a bronchus, when they are situated in the periphery of the lung, are productive of exquisite refinements of vocal fremitus. In this case the cavity acts as a resonating chamber for the amplification of the vocal vibrations. It is essential, however, that the cavity in these cases be free from fluid secretion, which may obstruct the bronchial orifice and consequently suppress vocal fremitus over its distribution. Bronchiectases, when peripherally distributed, cause similar exaggeration of tactile fremitus, bronchiectasis being the sole affection of the bronchi which is capable of increasing the intensity of vocal fremitus.

Diminished Vocal Fremitus.—Partial stenosis of the bronchi by tenacious secretions in the course of chronic bronchitis, or stenosis arising as a consequence of the lodgment of foreign bodies, or of bronchial compression by cicatricial bands, aneurysm, or extensive pericardial effusion, results in a diminution of vocal fremitus over the ultimate distribution of the affected tubes. When it is a case of partial occlusion by accumulated secretions, the fremitus is occasionally restored in its integrity after a severe paroxysm of cough.

Pleural thickening, sometimes noted at one apex as a result of chronic tonsillar infection, as well as in other portions of the pleural membrane as a sequel of pleural inflammation, is an occasional cause of diminution of vocal fremitus. That pleural thickening is not invariably attended by diminution of vocal fremitus was conclusively demonstrated by Wintrich in his experiments upon the cadaver. In his experiments he demonstrated that upon investing the exposed pleura with membranes prepared from the stomach or intestine there was no appreciable change in the intensity of the vocal vibrations when an assistant spoke into a tube which was inserted into the primary bronchus of the corresponding lung.

A very important group of cases, a group which is often quite confusing to the examiner at the bedside, is composed of certain cases of pulmonary infiltration and consolidation which, instead of yielding exaggeration of vocal fremitus upon palpation, exhibit an enfeeblement or abolition of tactile vibrations upon palpation. This condition is encountered in massive pneumonia, in which the terminal bronchi become occluded by fibrinous exudate with consequent suppression of vocal fremitus, and strongly simulating in this respect the picture of extensive pleural effusion. With less frequency, in the presence of carcinomatous infiltration of the lung, the neoplastic new productions develop in the interior of the bronchi, causing partial or complete occlusion of these passages, with consequent diminution or abolition of vocal fremitus over the area of the thorax corresponding to their distribution.

When the lung is separated from the thoracic wall by pleural effusion or by the accumulation of air or gas in the pleural cavity, vocal fremitus is enfeebled or is abolished. In the case of pleural effusion, it is possible for extensive adhesions between the visceral and the parietal pleura to transmit the vibrations across the effusion; and in these cases, also, the fremitus is ordinarily accentuated over the portion of the lung which is compressed into the superior part of the pleural cavity above the level of the effusion.

It is desirable, but not always possible, in connection with a pleural effusion to determine whether the presence of the fluid in the pleural cavity is the sole cause of diminution of the fremitus. Occasionally compression of the lung by the effusion results in compression and occlusion of the larger bronchi, a factor which plays an important part in suppressing the vocal fremitus. Moreover, in the presence of extensive effusions the thoracic wall is in a state of abnormal tension, which interferes with the transmission of vibrations arising within the thorax.

In dealing with pleural effusions, if the upper limit of the area of impaired vocal fremitus is carefully marked upon the thorax, the examiner is enabled to judge, during consecutive examinations, of the progress of the malady. In this disease, however, a sudden and pronounced descent of the level of the fluid is more apt to be due to relaxation of the diaphragm upon the affected side than to a sudden and pronounced resorption of the fluid.

In the case of encysted or loculated pleurisy, the examiner can occasionally delimit the effusion with fair accuracy by means of careful palpation with the ulnar border of the hand. However, Jaccoud, who has studied multilocular pleurisies with special reference to the variations in vocal fremitus, warns the examiner against the possibility of error in reaching a diagnosis of encysted pleurisy through the findings of palpation alone.

The accumulation of gas or air in the pleural cavity is attended by diminution of vocal fremitus over its distribution. In the case of pneumothorax limited to the apex of the pleural cavity, which yields a tympanitic note upon percussion, the enfeeblement of abolition of vocal fremitus over this area is of considerable aid in the differential diagnosis between a gaseous accumulation and a large tuberculous cavity in the apex of the lung.

In the interpretation of data obtained by the study of variations in the intensity of vocal fremitus in the various regions of the thorax, it is essential that the examiner be constantly upon the alert to detect any changes in the thoracic wall which might influence the intensity of the fremitus as appreciated by the palpating hand. In the presence of atrophy of one great pectoral muscle or of the muscles of the shoulder girdle, there is a corresponding exaggeration of vocal fremitus in the corresponding region, which might, during a casual examination, suggest the presence of apical consolidation. Circumscribed areas of edema of the thoracic wall and the presence of peripleuritic suppuration, on the other hand, are capable of producing a circumscribed diminution of vocal fremitus in the absence of bronchial or pleural disease. In the thorax which is the seat of extensive deformity from osseous or pulmonary disease deductions should be drawn from variations in vocal fremitus with caution and circumspection.

Limitations of Vocal Fremitus.—Aside from changes in the thoracic wall, the readiness with which vocal fremitus is elicited in diseased states of the thoracic viscera is influenced by a number of other factors, which frequently serve to limit the practical application of this method of examination. Thus, the early infiltrations of phthisis are not of sufficient extent to influence the intensity of the vocal vibrations, while the disseminated patches of bronchopneumonia also frequently fail to modify it to an appreciable degree.

The method finds its greatest field of usefulness in dealing with large consolidations and excavations, such as are encountered in lobar pneumonia and advanced phthisis. Similarly, in the presence of extensive pleural effusions and pneumothorax, the area of pathology is sufficiently extensive to render palpation of the vocal vibrations of distinct diagnostic value. In dealing with small infiltrations and consolidations vocal resonance, especially auscultation of the whispered voice, possesses greater diagnostic possibilities than does a study of variations in the intensity of vocal fremitus.

RHONCHAL FREMITUS

The term rhonchal fremitus, or bronchial fremitus, was first applied by Guttman to the vibrations which are generated by the passage of air in the respiratory passages through serum, mucus, pus, or blood in the bronchial tubes. The vibrations which are thus produced are readily appreciated by the palpating hand applied to the thorax. The lesions which are ordinarily responsible for the production of rhonchal fremitus are situated in the larger and medium-sized bronchial tubes when it is a question of the passage of air through tenaceous secretions; but the lesions which are provocative of the fremitus also not infrequently assume the form of tuberculous cavities containing fluid with a bronchial communication situated below the level of the fluid, through which air descends during inspiration and creates bubbling which is transmitted to the thoracic surface in the form of rhonchal fremitus. Hence, it follows that rhonchal fremitus may possess a variable diagnostic and prognostic significance depending upon the mechanism of its production and the region of the thoracic surface upon which it is encountered.

The rhonchal fremitus which originates in the larger and medium-sized bronchi in the course of chronic bronchitis and bronchial asthma, and which is not of especially grave prognostic import, is encountered in the upper sternal, lower infraclavicular, and upper mammary regions. Rhonchal fremitus encountered in these regions has a very considerable intensity, and it is commonly felt over a considerable area of the thoracic surface. Rhonchal fremitus which is engendered in a tuberculous cavity, on the contrary, is ordinarily encountered in the supraclavicular and infraclavicular regions, corresponding to the apices of the lungs; it has not the intensity of the fremitus generated in the larger bronchi; and it is of grave prognostic import. In palpating in these regions, the examiner will not infrequently encounter crepitations which are due to the contractions of the great pectoral muscle, particularly when dealing with robust subjects, and which may easily be mistaken for rhonchal fremitus.

Similarly, rhonchal fremitus is apt to be confused with pleural friction fremitus. In this connection it is to be remembered that in dealing with pleural friction fremitus, the latter is exaggerated upon compression of the intercostal spaces; that pleural friction fremitus is ordinarily attended by pain which is exaggerated by pressure exerted with the finger tips in the intercostal spaces; and that rhonchal fremitus arising in the bronchial tubes is frequently abolished by the act of coughing, whereas pleural friction fremitus is not influenced by this act.

Rhonchal fremitus is transmitted to the thoracic surface with the maximum intensity in women and children, in whom the thoracic wall is thin and elastic, and in the emaciated patient; and due allowance should be made for the relative thickness of the thoracic parietes in interpreting the intensity of rhonchal fremitus in any case in which it is encountered.

PLEURAL FRICTION FREMITUS

In the normal subject the visceral and the parietal pleura are moistened by a small quantity of serous fluid which enables the membranes to glide over each other during the respiratory movements of the lungs and the thoracic walls without the production of sound. In the presence of pleural inflammation, however, the surface of the membrane becomes invested by a coating of fibrinous exudate of varying thickness, by virtue of which there is produced during the respiratory movements an audible rubbing sound, the pleural friction sound. Upon palpation of the thorax over the region of production of the pleural friction sound there is frequently encountered a palpable vibration or fremitus, which Guttman first designated as pleural friction fremitus. The tactile impression which one obtains is that of grating, which is variable in intensity in different cases and which is dependent upon the respiratory excursions for its generation. It is most frequently encountered in the infraaxillary region and the lower mammary region; and it is most frequently generated in a vertical direction, with less frequency in an oblique direction, and still less frequently in a horizontal direction.

The fremitus is appreciated most frequently during inspiration, with less frequency during inspiration and expiration, and very rarely during expiration alone. It is not infrequent that the intensity of the fremitus is so slight that it is not perceived during tranquil respiration, but is brought to the fore by a series of deep inspirations. Also, it is not infrequent for the fremitus to disappear after a series of deep inspirations, probably by reason of the smoothing out upon the pleural surface of the rugosities of exudate which called the fremitus into being. At any rate, the fremitus frequently reappears upon deep inspiration following a period of tranquil respiration on the part of the patient. The intensity of the fremitus can be reinforced by pressure upon the intercostal spaces in the area of its production, a procedure which is attended by exaggeration of the thoracic pain.

Pleural friction fremitus can sometimes be elicited by having the subject hold the breath at the end of inspiration and elevate the arms as far as possible, thus causing the parietal pleura to move upward upon the fixed visceral pleura.

Pleural friction fremitus is encountered in the presence of acute fibrinous pleurisy and during the incipient stage of serofibrinous pleurisy. In the latter disease the fremitus is present prior to the development of the effusion; it disappears with the free establishment of effusion; and it very frequently recurs with partial or complete absorption or evacuation of the effusion.

TUSSILE FREMITUS

Tussile or tussive fremitus is a palpable thoracic vibration which is produced upon coughing. It is not invariably present; it does not possess great value in physical examination; and it is not frequently elicited during the routine physical examination. It is to be employed in dealing with patients who are subject to aphonia from any cause, when it is not possible to elicit vocal fremitus.

SUCCUSSION FREMITUS

Succussion fremitus is a palpable thoracic vibration which is produced when a patient whose pleural cavity contains air and fluid is suddenly jarred or shaken. Under these circumstances the impact of the fluid is felt against the palpating hand as a vibration.

Occasionally extensive pulmonary cavities which are situated in the peripheral portions of the lung give the vibration, if they contain air and fluid. However, the physical conditions are not ideal for the elicitation of this sign from pulmonary cavities; and it is much more frequently encountered in hydro-, hemo-, or pyo-pneumothorax.

HYDATID FREMITUS

In the presence of hydatid cyst of the lung with visible protrusion of the thoracic wall, it is occasionally possible to elicit hydatid fremitus by placing the fingers, widely separated upon the bulging area and percussing lightly upon one of the fingers. Under these circumstances a vibration which is caused by the impact of the daughter cysts is occasionally appreciated by the fingers.

CREPITATION

In cases of surgical emphysema, when the subcutaneous tissues of the thorax contain small beads of air, a fine crepitation is often demonstrable upon palpation of the affected area with the finger-tips. Air may gain access to the subcutaneous tissues of the thorax as a result of trauma or operation upon the neck or chest, or from the rupture of dilated infundibula in hypertrophic emphysema.

LOCAL TENDERNESS

In the presence of disease of the pleura and in disease of the lung complicated with pleurisy, palpation frequently reveals areas of local tenderness upon the thoracic surface. The pain in these cases is elicited and is also defined by finger-tip palpation of the intercostal spaces. Such localized tenderness is suggestive, in the first place, of acute fibrinous pleurisy; and with less constancy it points to intercostal neuralgia, pleurodynia, fracture or caries of the ribs, deeply-seated disease of the lung, or disease of abdominal organs.

Bau and Bouillaud explain the local sensibility of certain intercostal spaces in connection with acute fibrinous pleurisy on the basis of a neuritis of the corresponding intercostal nerves. They call attention to the fact that the intercostal nerves in the posterior third of their course are in direct relation with the under surface of the pleural membrane, and Bau has demonstrated microscopically that neuritis occurs in connection with this pain in association with acute fibrinous pleurisy and pleuro-pneumonia.

The fact that the pleural pain is manifested not upon the posterior thoracic surface, but upon the lateral and anterior aspects of the chest is explained by the fact that irritation of the nerve trunk is transmitted throughout its terminal expansions. The predominance of the pleural sensibility in the sixth and seventh intercostal spaces is due to the fact that this region of the thorax is more mobile than other portions, and the extreme mobility of these interspaces intensifies the pain upon pressure with the finger tips.

Malloizel, who has studied pleural pain with special reference to interlobar pleurisies, was able in many instances of this disease to elicit tenderness which corresponded accurately to the course of the interlobar fissure, commencing in the neighborhood of the third dorsal vertebra, and radiating toward the lateral and anterior thoracic regions along the course of the interlobar fissure in question.

While as a general rule even extensive inflammatory disease of the lung is unattended by pain upon pressure exerted in the intercostal spaces, in pleural inflammation of whatever intensity this local tenderness is almost invariably present. However, in the case of lobar pneumonia, Hutinel and Paisseau have described a submammary painful point in adults, whereas in infants and children the pain is commonly referred to the right iliac region, simulating that of acute appendicitis in this class of patients.

The pain of intercostal neuralgia is not infrequently limited to a single intercostal space, but in this space it commonly involves the entire area from the vertebral column to the sternum. Moreover, it is attended by the three painful points of Valleix, pressure upon which accentuates the pain. These points correspond to the points of emergence of the cutaneous filaments of the intercostal nerves. One of these points is situated in the posterior extremity of the intercostal space close to the vertebral column; another is situated upon the lateral thoracic wall equi-distant from the vertebral column and the sternum; while the third is situated upon the anterior thoracic surface adjacent to the lateral sternal border. In addition to the presence of these painful points, the pain of intercostal neuralgia is paroxysmal in its character.

Neuralgia of the phrenic nerve causes characteristic tenderness of the thorax. In this condition pain is elicited upon the exertion of pressure upon the antero-lateral thoracic wall at the insertions of the digitations of the diaphragm into the seventh, eighth, ninth, and tenth ribs; upon the insertion of the muscle to the twelfth rib adjacent to the vertebral column; and upon the spinous processes of the third and fourth cervical vertebræ, which correspond to the origin of the cervical plexus. While neuralgia of this nerve occasionally develops without apparent cause, in many instances the underlying factor is a diaphragmatic pleurisy or a pericardial effusion.

The pain of pleurodynia is most frequently observed upon the left side of the thorax, involving usually the intercostal muscles, and with less frequency the pectoralis major and the serratus magnus. Pressure exerted with the finger-tips, movements of the thoracic wall during respiration, and bending and turning of the trunk intensify the pain. The distribution of the pain is more diffuse than is the case with intercostal neuralgia, and the pain is constant and not paroxysmal as is the case with neuralgia of the intercostal nerves. When pleurodynia involves the pectoralis major muscle the pain is greatly aggravated when the examiner compresses the body of the muscle between the fingers.

In periostitis or caries of a rib, the tenderness is limited to the rib in question, and usually to a single portion of the rib, while compression of the adjacent intercostal spaces is almost devoid of pain. Occasionally there is local bulging and discoloration of the integument over the site of the lesion. Localized pain upon pressure upon a rib is frequently encountered as a result of fracture of a rib.

Local tenderness due to peripleuritic abscess is commonly attended by a local bulging of the thoracic wall, and in the later stages of the condition, when perforation of the chest wall is imminent, there is local bulging and discoloration of the integument.

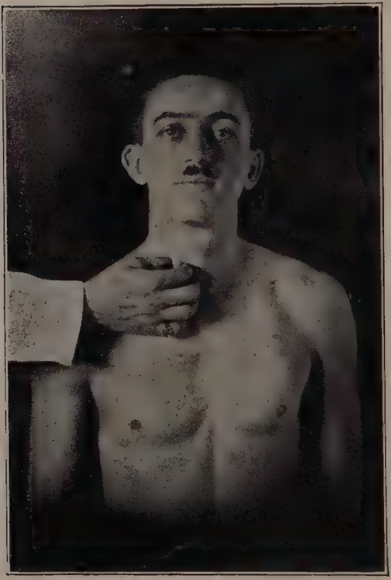


Fig. 138.—Palpation of sternomastoid.

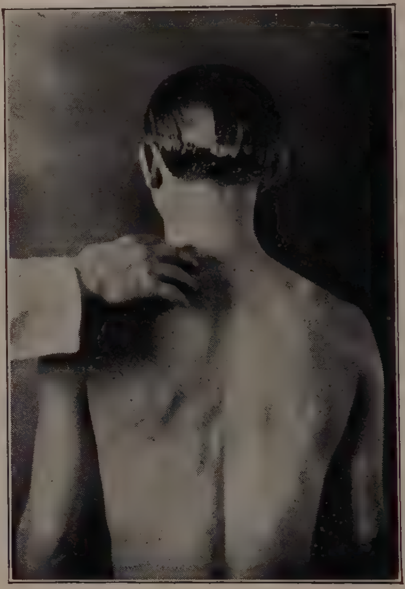


Fig. 139.—Palpation of trapezius.

Skin and Subcutaneous Tissues.—Atrophic changes affecting the skin and subcutaneous tissues of the thorax follow destructive lesions of the nerves supplying these parts. A more important cause of atrophic changes in these structures, as shown by Pottenger, is a quiescent or healed tuberculous or nonspecific lesion of the corresponding lung or pleura. The wasting of the tissues is most clearly in evidence in the episternal, supraclavicular and infraclavicular regions anteriorly, and in the supraspinous and upper interscapular regions posteriorly, where it produces a characteristic flattening of the upper thorax, with undue prominence of the bony structures. While usually evident upon inspection of the thorax, the extent and degree of atrophy are more readily made out upon palpation with the finger tips.

In the presence of an active lesion of the underlying pulmonary tissues, on the contrary, the muscles are found to be in a state of reflex motor spasm in regions corresponding accurately to the distribution of the trophic nerve paths from the area of pulmonary disease. The spasm is best elicited by light touch palpation, the examiner placing the finger tips lightly upon the integument over the muscles and alternately pressing and releasing the pressure. Under these circumstances a sensation of increased resistance upon the part of the subjacent muscles is appreciated. The increased tone may also be appreciated by gently picking up the muscle in regions where it is accessible, and rolling it between the thumb and finger. (Pottenger.)

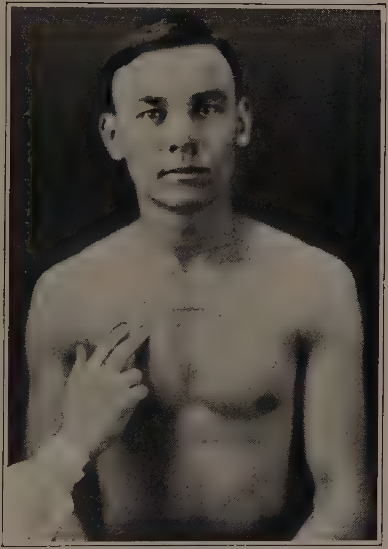


Fig. 140.—Palpation of the intercostal spaces.

The muscles most frequently involved are those of the upper thorax and shoulder girdle, notably the sternomastoids, scaleni, upper fibers of the pectoralis major, trapezius and levator anguli scapuli.

THE INTERCOSTAL SPACES

In addition to the elicitation of local tenderness, palpation of the interspaces is employed in the estimation of their relative width in cases of pleurisy with effusion, chronic ulcerative phthisis, and in unilateral and local deformities of the thorax due to pulmonary fibrosis or extensive bronchiectasis. In the presence of pleurisy with effusion, the consolidations of lobar pneumonia and phthisis, and in cases of excessive pleural thickening, as well as in fully developed cases of hypertrophic emphysema, finger-tip palpation of the intercostal spaces yields a sensation of increased resistance.

THE RIBS AND STERNUM

In addition to the elicitation of tenderness in the presence of inflammation and caries of the ribs, palpation of these structures in syphilitic subjects occasionally reveals the presence of gummatous nodules. In rickets the rachitic rosary is often present in the form of a series of palpable or even visible nodes occurring in series upon either side of the thorax at the junction of the ribs with their costal cartilages.

Palpation of the sternum is serviceable in the detection of minor variations in the prominence of the angulus Ludovici, and also sometimes reveals the presence of caries of this bone. Painful enlargement of the sternoclavicular articulation is more common in connection with gonorrheal rheumatism than in any other disease. Prominence of the superior portion of the sternal region, attended by pain of a dull, boring character, is significant of aneurysm of the aortic arch.

FLUCTUATION

Fluctuation is not ordinarily demonstrable in connection with disease of the thoracic viscera, on account of the rigidity of the thoracic wall. Pleural effusion, if it is very extensive and is attended by marked widening of the intercostal spaces, occasionally yields fluctuation upon tapping the thoracic wall while the fingers of the opposite hand are pressed firmly into the intercostal space. This fluctuation is more frequently demonstrable in the case of children with empyema.

In cases of empyema necessitatis, in which the accumulation of purulent material is extensive and rupture is imminent, pitting upon pressure is readily obtained, and fluctuation is not infrequent. It is elicited by placing the palm of the palpating hand over the protruding area and rather forcibly striking the opposite side of the thorax with the free hand, a procedure which is not devoid of danger. In most cases of empyema necessitatis the local bulging may be made to disappear by progressive pressure exerted upon the tumor by the palm of the hand. Upon removal of the pressure the tumor resumes its former dimensions. Not infrequently the size of the protrusion caused by empyema necessitatis is variable, depending upon the depth of the respirations. Fluctuation is occasionally demonstrable in the presence of abscess of the pectoral wall, whether due to disease of the thoracic musculature, the ribs, or caries of the bodies of the dorsal vertebræ. Aneurysm of the aortic arch, with erosion of the sternum, presents an expansile, fluctuating tumor, above the base of the heart.

LOCAL PULSATION

Palpation is serviceable in confirming the findings of inspection as to pulsation of various regions of the thoracic surface; for detecting minor degrees of thoracic pulsation which may have escaped detection during inspection; and to gauge the force or lifting power of a thoracic pulsation.

In empyema, when this involves the left pleural cavity, the impact of the heart is communicated to the fluid during cardiac systole, creating thus a systolic pulsation in the lower left anterolateral thoracic region. Frantzel has described a similar systolic pulsation which was transmitted to a purely serous pleural effusion, and Eichhorst has encountered in the child similar pulsations in serous effusions of the right pleura, in this instance localized to the right lower anterolateral thoracic region. Similarly, when the lingula of the left lung which overlies the heart in the left mammary region becomes the seat of dense consolidation in pneumonia or is invaded by dense carcinomatous infiltration, there is generated a systolic pulsation or pulsion of this region of the thoracic wall with each ventricular systole.

In the presence of empyema necessitatis of the left anterolateral thoracic wall, there is produced, just as in the case of aortic aneurysm, a systolic pulsation which is expansile in character, occurring in all directions. This expansile pulsation of the protrusion may be determined with ease by applying the fingers widely separated to the various walls of the sac simultaneously, whereupon the fingers will be moved apart with each systole of the heart which is transmitted to the contained fluid.

In the differentiation of such a pulsating empyema necessitatis from aneurysm Müller calls attention to the fact that empyema is situated in the lower left antero-lateral region of the thorax, whereas aneurysm is localized in the upper and right portion of the thorax; that in the case of empyema, progressive, gentle pressure exerted upon the protrusion will cause its reduction by evacuation of the purulent contents into the pleural cavity through the fistulous communication between the pus sac and this cavity; that in empyema the zone of dullness extends well beyond the limits of the tumor; and that in empyema no systolic bruit is audible as is the case with aneurysm.

A peripleuritic abscess overlying the ventricle of the heart is attended by a systolic pulsation in this area; but in this instance, as the purulent accumulation is enclosed in a cavity with relatively dense walls, the pulsation is manifested as a simple raising and recession and is not expansile.

A palpable pulsation occurring over a relatively broad area upon the left side of the posterior wall of the thorax below the angle of the scapula, is occasionally noted in connection with an extensive tuberculous excavation of the left lung, when this cavity is filled with fluid, to which the impact of the heart is transmitted during systole.

PALPATION OF THE CARDIAC IMPULSE

The cardiac impulse is not always visible and when one cannot be guided by the visible impulse an attempt should be made to locate it by the sense of touch. Beginning at the fifth intracostal space, about 8 cm. from the midsternal line, move the tips of the fingers, first along the fifth intracostal space to the left and right, then along the sixth and seventh, and finally along the fourth, instructing the patient to lean forward and slightly to the left. After locating the impulse, the hand should be placed flat on the thorax with the tips of the first three fingers a little beyond the maximal visible impulse or approximately where the impulse has been located by the finger tips. After feeling with the whole hand, greater accuracy may be obtained by palpating with the first and second fingers of the right hand, first together and then separated.

The rate, force, and rhythm should be carefully noted and when abnormalities appear should be compared with the radial pulse. In cases of auricular fibrillation, pulsations may be detected at the apex which do not appear at the wrist.

Absence of apex impulse may occur in very obese individuals and in women with large and pendulous breasts. Pulmonary

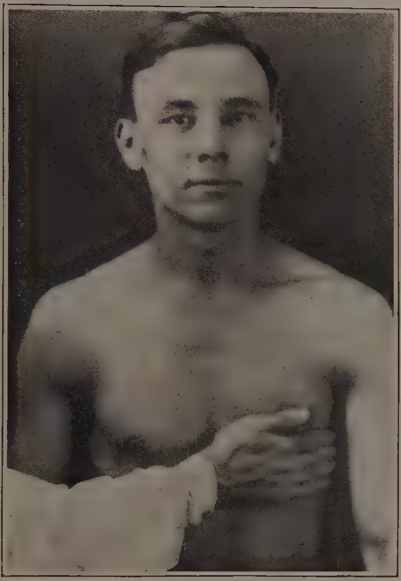


Fig. 141.—Palpation of cardiac impulse (first maneuver).

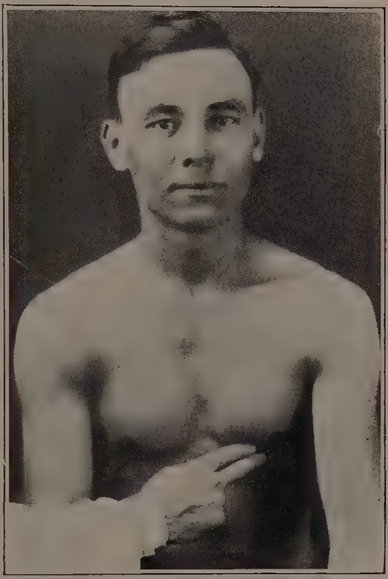


Fig. 142.—Palpation of cardiac impulse (second maneuver).

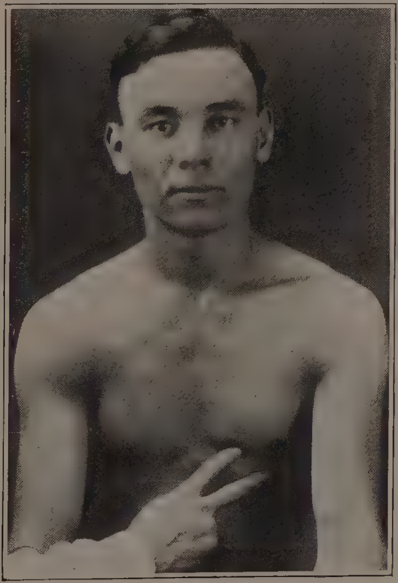


Fig. 143.—Palpation of cardiac impulse (third maneuver).

emphysema may also obscure the cardiac impulse. The heart muscle may not contract with sufficient force to produce a palpable impulse. Fluid in the pericardial sac may obliterate the cardiac impulse.

Other pulsations on the thorax should be palpated and their pulsations compared with the cardiac impulse.

The impulse of thoracic aneurysm may be preceptible later than the apex impulse.

THRILLS

Upon light palpation of the precordia of a patient who is the subject of valvular disease of the heart, aneurysm, or anemia, a local vibration of the thoracic wall, or *thrill*, is apt to be detected at one or more points. The sensation which is appreciated during palpation is somewhat similar to that which is experienced upon placing the hand upon the throat of a purring cat, hence the term *frémissement cataire,* occasionally employed to describe it.

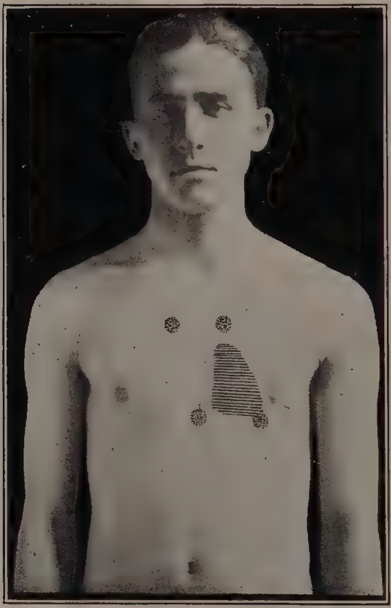


Fig. 144-*A*.—Sites of palpable thrills and pericardial friction fremitus.

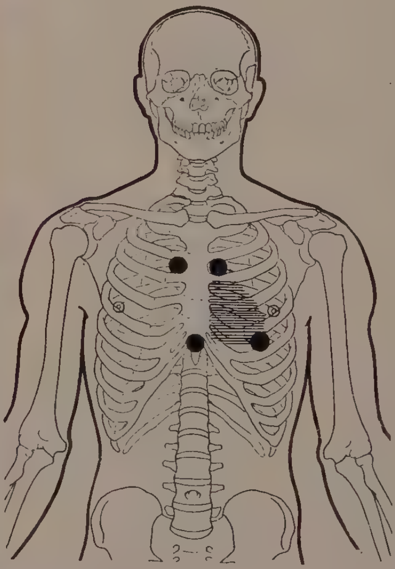


Fig. 144-*B*.—Sites of palpable thrills and pericardial friction fremitus.

Thrills are endocardiac, vascular or hemic in origin; and in point of time they are presystolic, systolic or diastolic, as they occur prior to, during, or after ventricular systole.

A thrill of endocardiac origin is generated by the same mechanism which is responsible for the production of an endocardiac murmur; namely, a narrowing of an orifice through which the blood stream is propelled into a larger chamber of the heart. As long as the normal quota of blood of normal density flows through normal orifices, no abnormal vibration is generated; but when the lumen at one point is diminished, the blood which passes through this stenotic orifice into the wider chamber beyond is thrown into eddies, creating the so-called "fluid veins," the vibrations of which are conveyed to the precordia as a tactile vibration, or thrill, which is the tactile equivalent of a murmur.

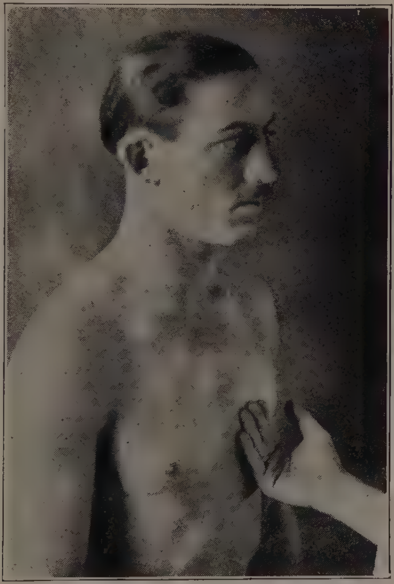


Fig. 145.—Palpation for thrill.

As thrills of endocardiac origin are confined to limited areas of the precordia, they are most surely detected in the first instance by light palpation with the palm of the hand, after which they may be more definitely defined by finger-tip palpation. Firm pressure with the palm frequently suffices to obliterate a thrill. Certain clinicians prefer to palpate for thrills with the dorsal aspect of the fingers.

The intensity of a thrill is very variable, as the vibration is very prone to disappear in the case of a feebly acting heart, and to reappear with improvement in the state of the myocardium. The quality of a thrill varies with the rate of the vibrations which call the thrill into being, rapid vibrations of the blood stream producing fine thrills and murmurs of high pitch, whereas less rapid vibrations result in coarse thrills and murmurs of lower pitch.

In general, a systolic thrill at the base of the heart is indicative of aortic aneurysm, exophthalmic goiter, or structural changes in the aortic or pulmonary valves. The thrill of aortic aneurysm is commonly localized in the second and third interspaces to the right of the sternum; that of aortic stenosis is detected in the second right intercostal space adjacent to the sternal border; while the thrills of pulmonary stenosis and exophthalmic goiter are palpable in the second left interspace adjacent to the border of the sternum.

Diastolic thrills at the aortic and pulmonary areas occasionally accompany regurgitant lesions of the aortic and pulmonary valves; but thrills are demonstrable with greater constancy in the presence of stenotic than of regurgitant lesions.

A presystolic thrill localized over the cardiac apex in the fifth left intercostal space is a very good sign of mitral stenosis. However, a similar thrill has been found to accompany the Flint murmur of aortic regurgitation in this area. Occasionally is a systolic thrill to be detected over the apex in the course of mitral regurgitation.

A presystolic thrill is occasionally to be elicited over the tricuspid area in the presence of tricuspid stenosis; while a systolic thrill in the same area is occasionally to be detected in tricuspid regurgitation.

In the interpretation of precordial vibrations the examiner should remember that in the presence of moderate cardiac hypertrophy in a subject with a thin chest wall, or in the presence of fibroid retraction of the anterior border of the left lung in the anemic subject, the impact of the conus arteriosus of the right ventricle against the thoracic wall may generate a physiologic thrill in the absence of valvular disease.

VALVE SHOCK

Valve shock is due to the impact of closure of the valves of the heart, and it can be appreciated when the palm of the hand is applied flatly over the valve area in question. The shock is occasionally to be elicited over the areas of the auriculoventricular valves, but not with the same constancy as is the case with the aortic and pulmonary valves. In either case the thickness of the thoracic wall, the state of the superimposed pulmonary tissues, and the state of the myocardium and arteries exert a striking influence upon the intensity of the shock as appreciated by the palpating hand.

Valve shock elicited over the auriculoventricular valves is systolic in time, corresponding to the closure of the cusps of these valves; whereas the shock over the aortic and pulmonary valves occurs during the first portion of ventricular diastole, at the instant of closure of the cusps of the semilunar valves.

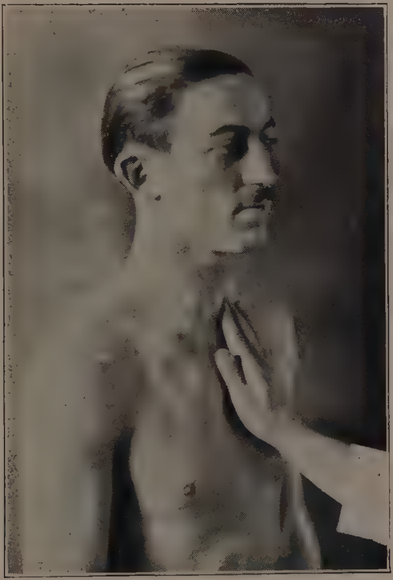


Fig. 146.—Palpation of valve shock at base.

Accentuation of the shock over the pulmonary area is noted in the presence of right ventricular hypertrophy, and when the pressure in the pulmonary circulation is raised as a result of valvular lesions of the left side of the heart or obstructive diseases of the lungs.

The aortic shock is accentuated in the presence of left ventricular hypertrophy, and in arteriosclerosis and chronic renal disease, conditions which are attended by hypertension of the greater circulation.

PERICARDIAL FRICTION FREMITUS

Friction fremitus of pericardial origin is generated during the evolution and course of acute fibrinous pericarditis or during pericarditis with effusion. In the latter disease the fremitus is prone to disappear with the gradual accumulation of the effusion, which separates the layers of the pericardium, though it is not uncommon for the fremitus to persist at the base of the heart during the height of the effusion.

Pericardial friction fremitus is usually to be detected in the second, third and fourth interspaces along the left border of the sternum; it does not extend beyond the limits of the precordia; it is frequently attended by moderate pain; and the pain is accentuated by pressure exerted over the lower portion of the sternum.

Pleuropericardial friction fremitus is elicited over the distribution of the lappet of lung which is in apposition with the right ventricle, or over the portion of the pericardium which is in intimate relation with the anterior thoracic wall in the incisura cardiaca. In the first case the fremitus is abolished with the recession of the anterior pulmonary border during forced expiration, whereas in the second instance the fremitus is enfeebled or abolished during forced inspiration, when the anterior pulmonary border intervenes between the pericardium and the costal pleura.

CHAPTER XIX

PALPATION OF THE ABDOMEN

Technic.—During palpation of the abdomen the patient should assume the dorsal decubitus with the head slightly elevated by a small pillow and the knees drawn up and supported by a pillow, which relieves the tension of the abdominal wall. In bed-ridden subjects a similar state of abdominal relaxation may be attained by propping the patient's shoulders up with pillows and drawing up the knees and supporting them. Under certain circumstances it is desirable to palpate the abdomen with the patient in the knee-chest position or when standing.

The patient having been placed in a natural and unconstrained attitude, he should be directed to refrain from the natural tendency to hold the breath during the examination.

The right-handed examiner stands or sits on the subject's right. With the left-handed examiner the procedure is reversed. The hands of the examiner should be warm, as a cold hand applied to the surface of the bare abdomen will cause a local muscular rigidity which combats and frustrates the object of the examination. The examining hand should be applied very gently to the abdominal surface with the palm down and the fingers extended, avoiding any sudden pressure or punching movements. Within a few moments it is possible in most cases to secure sufficient relaxation of the muscles to permit the hand to depress the wall with a gently rolling movement.

As it is necessary to keep the abdomen relaxed, it is wise to follow a routine palpation which will be least likely to cause pain; therefore it is wise to palpate areas ordinarily not painful before attempting palpation of areas usually sensitive or frequently diseased.

After the routine palpation the examiner may go back to a suspicious area and palpate more thoroughly, using additional methods of palpation as seem necessary. For this reason a routine palpation will be described followed by notes on special palpation of various organs.

The hand should first be moved over the surface of the abdomen firmly but gently, noting any unusual resistance or tendency to contraction of muscles due to abnormal sensitiveness or "ticklish" points. After the subject has been accustomed to the feel of the examiner's hand a systematic palpation of the abdomen may be begun.

THE SPLEEN

Beginning with the upper left quadrant one first palpates for the spleen. Place the right hand at right angles to the left costal margin in the epigastric angle and the left hand over the chest wall just above the costal margin with its long axis at right angles to the palpating fingers.

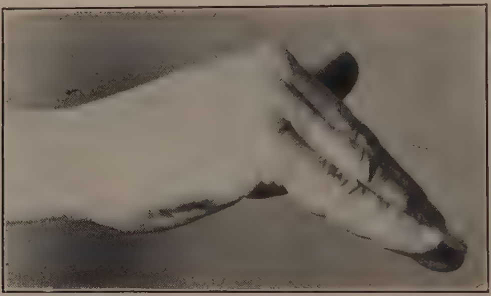


Fig. 147.—Correct method of flexing the hand for abdominal palpation.

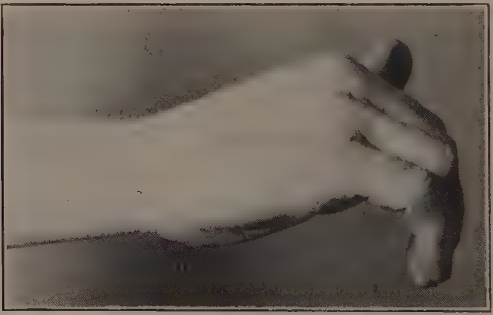


Fig. 148.—Incorrect method of flexing the hand for abdominal palpation.

The subject is then instructed to take a moderately deep breath. At the end of inspiration the left hand pulls down the skin of the thorax while the right is forced gently but firmly upward under the edge of the ribs. This procedure is repeated several times, each time using more force with the right hand, bending the fingers at the metacarpophalangeal joint only, this avoids a clawing motion which tends to produce muscular contraction. To bring the spleen nearer to the anterior abdominal wall, the subject may lie upon the left arm, so that the arm crosses the back at about the ninth rib. Another method is to have the patient turn on the right side or in the right diagonal position. Normally, the spleen cannot be felt, but when enlarged it is often quite easily felt, and usually exhibits a notch about the middle of the left border.

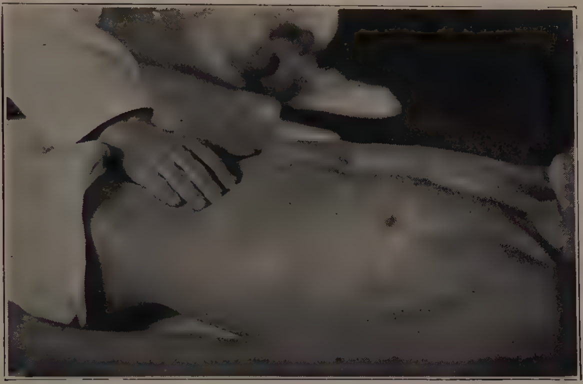


Fig. 149.—Palpation of the abdomen. Palm applied lightly to integument.

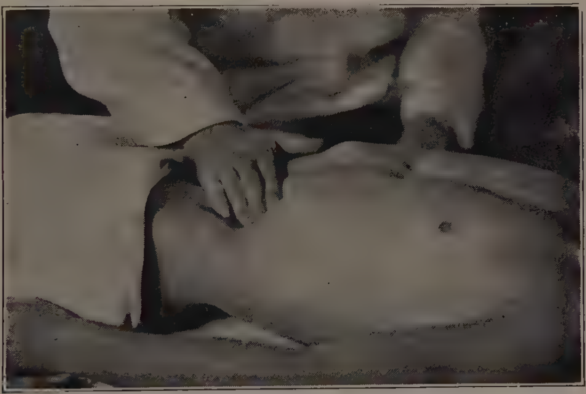


Fig. 150.—Palpation of the abdomen. Depressing the wall in various regions with the fingers.

During palpation of the spleen the examiner should take note of its form, dimensions, consistency, sensibility, mobility, the state of the surface, the presence of splenic friction fremitus or of pulsations, and the site in which the organ is encountered.

The hypertrophied spleen may preserve its normal form, or its contour may be markedly distorted from the normal, the contour of the organ in the two instances offering some indications as to the cause of the hypertrophy. The moderate acute enlargements of the organ are quite constantly attended by the preservation of the normal contour of the organ, as are also the chronic enlargements due to diseases of the blood or to chronic valvular disease of the heart. In the case of splenic enlargement due to malignant disease, to cystic disease, or to abscess of the gland, however, the normal contour of the organ is distorted and one or more protrusions may sometimes be palpated upon its surface.

The *dimensions* of the hypertrophied spleen present numerous gradations from the moderate acute hypertrophies which are scarcely palpable to the immense leukemic spleen which may occupy the major portion of the abdominal cavity. Hyrtl encountered at autopsy an enlarged and indurated spleen which filled the abdominal cavity and which had created a pressure perforation of the left iliac bone.

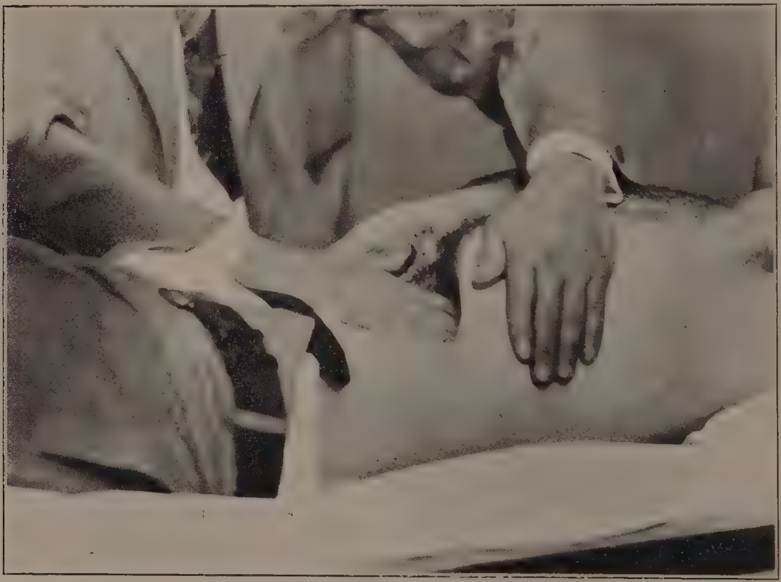


Fig. 151.—Palpation of the spleen.

The *consistence* of the enlarged spleen varies with the cause of the hypertrophy, the volume of the organ, and, with the duration of the enlargement. Acute transient hypertrophies of the spleen arising during the course of the acute infections are not attended by any notable alteration in the consistence of the spleen, and such hypertrophies are very frequently not palpable for this reason. In the immense hypertrophies of leucemia and splenic anemia, on the contrary, the organ is hard and occasionally of almost cartilaginous consistence. The amyloid spleen possesses a ligneous consistence in its advanced stages. Splenic tumors may present multiple hard masses, or a single large protrusion which may yield fluctuation. Barbieri has elicited fluctuation in the case of a large splenic abscess which was afterward treated surgically. Similarly, in the case of hydatid cyst of the spleen fluctuation may occasionally be demonstrated, as well as hydatid fremitus upon palpatory percussion.

Tenderness upon palpation of the spleen is as a rule only elicited upon deep pressure, and in this case it is rather to be attributed to stretching of the capsule of the organ than to irritation of the splenic parenchyma. In perisplenitis attending acute splenitis or the enlarged spleen of acute splenic hyperemia tenderness is most readily elicited. Splenic tumors rarely exhibit tenderness with the exception of splenic carcinoma. *Signorelli's spleen point*, to which pain is referred in splenic disease is situated immediately below the junction of the fifth left costal cartilage and the midclavicular line.

Splenic enlargements exhibit rather a remarkable range of *mobility*. Like the liver, the spleen exhibits respiratory mobility, the enlarged organ descending with each inspiration, only to remount during expiration. In the upright attitude splenic enlargements occupy a lower position in the abdominal cavity than obtains during the dorsal decubitus. Upon assuming the right lateral decubitus, an enlarged spleen deviates toward the right side of the abdominal cavity and at the same time downward; while upon assuming the left lateral decubitus the opposite range of mobility is noted. The enlarged spleen is readily mobile upon manual manipulation.

A further element in splenic mobility consists in relaxation of the splenic ligaments, particularly in the case of multiparæ, whereby even in the case of a spleen of normal dimensions a condition of "floating spleen" is frequently created. Under these circumstances the organ may be encountered in the pelvis and Morgagni and Ruysch once found the viscus displaced into an inguinal hernia. The displaced spleen is ordinarily recognized by the palpable hilum of the organ, in which position the pulsations of the splenic vessels are occasionally detected. A movable spleen may occupy a position superjacent to the abdominal aorta and have the pulsations of this vessel communicated to it in the form of a systolic rise and diastolic fall of the tumor. Placing the patient in the genupectoral posture causes the spleen to fall away from the vessel with the consequent suppression of the pseudo-pulsation.

The *surface* of the enlarged spleen may be smooth and uniform, or it may be irregular and embossed. The acutely hypertrophied spleen and the spleen of the earlier stages of chronic passive congestion present a smooth and uniform surface; while the spleen which is the site of infarction, abscess, echinococcus cyst, gummata, or of carcinomatous infiltration presents an irregular surface with nodules or bosses which may or may not be umbilicated.

Friction fremitus is occasionally appreciated upon palpation of the abdomen over the spleen which is the seat of perisplenitis. This alteration of the splenic surface is much more frequently translated in the form of an audible friction sound, however, than it is demonstrable as a friction fremitus. Occasionally in the case of hydatid disease of the spleen one can elicit hydatid fremitus over the organ; but this sign is not present in the case of hydatid cyst of the spleen with the same frequency with which it is encountered over the lung or the liver.

Theoretically the systolic pulsation of the liver attending advanced tricuspid regurgitation should be appreciable over the spleen; but clinically it is the exception to encounter such a systolic pulsation. Pryor and Drasche have, however, described pulsations of the spleen in subjects suffering with aortic insufficiency.

Aside from the floating or wandering spleen which develops as a result of relaxation of the suspensory ligaments of the organ, the spleen may be displaced in a vertical direction by alterations in the proportions existing between the pressures which are exerted upon the superior and inferior surfaces of the diaphragm in the presence of diseases of the thoracic and the abdominal viscera.

Downward displacement of the spleen may be caused by increased intrathoracic tension from hypertrophic emphysema, left-sided pneumothorax or pleural effusion, massive pneumonia of the left lung, an extensive pericardial effusion, or a thoracic neoplasm. The spleen also occupies a lower position than normal in Glenard's disease.

Upward displacement of the spleen occurs when it is pressed upon by ascites, tympanites, or large abdominal tumor. Fibroid retraction of the left lung or paralysis of the diaphragm will likewise cause it to occupy a higher level in the abdominal cavity than is normal.

THE KIDNEYS

After palpating the splenic area the right hand is turned so that the long axis of the hand is at right angles to the long axis of the body. The ends of the fingers should rest at about the attachment of the tenth rib. This should be accomplished without removing the hand from the abdominal wall. The left hand is then moved backward to the thick muscular region to the left of the last dorsal vertebra. The hands are now in a proper position to palpate the left kidney.

The subject is told to take a deep breath and at the end of inspiration an attempt is made to bring the fingers of the right and left hands together. All movements of the right hand should be from the metacarpophalangeal joint. Never palpate the abdomen with crooked fingers.

The kidney being at the lowest level, at the end of inspiration it can be palpated if at all palpable. Several attempts with increasing pressure are often necessary. The normal kidney occupying a normal position is palpable with difficulty and frequently is not palpable. If during these maneuvers the inferior pole of the kidney is felt at the end of inspiration but slides into place during expiration the condition constitutes a movable kidney.

Three degrees of movable kidney are recognized. In the first degree, only the inferior pole of the kidney is palpable; in the second degree the inferior half of the kidney is palpable; while in the third degree of movable kidney the entire anterior face of the organ is palpable.

If, on the contrary, the kidney fails to glide back into its normal position during expiration, if during this period the entire kidney remains palpable and can be pushed about in the abdominal cavity, the condition is termed *displaced kidney*, or *floating kidney*. The right kidney is frequently movable in girls and women, particularly in subjects with moderate enteroptosis; with less frequency is the kidney movable in the male subject.

A movable kidney, a displaced kidney, and the kidney which is enlarged as a result of hydronephrosis, neoplasm, pyelonephrosis or surgical kidney is readily palpable and occasionally yields fluctuation under proper conditions.

When upon palpation of the kidneys the examiner encounters an abnormality in the dimensions or contour of the kidneys, he should proceed to make a detailed study of the sensibility, the form, and the volume of the kidney, its limits in respect to the adjacent abdominal viscera, its range of mobility, the state of its surface, and its consistence.

The *sensibility* of the kidney is variable both in the normal subject and in pathologic states of the organ. The subjective sensation which is experienced upon compression of the normal organ is dull and aching, somewhat analogous to the sensation which is experienced upon moderate compression of the testicle. In the presence of pathologic changes in the organ the sensibility ranges from this dull pain to very lively, excruciating pain. Frerichs calls attention to the fact that the left kidney is normally more sensitive to pressure than is the right kidney, probably as a result of its more superficial position, with the result that a greater degree of pressure is exerted upon this kidney during the routine palpation of the kidneys. In renal disease tenderness may be elicited upon pressure with the finger tips upon the anterior abdominal wall two inches below the costal arch and slightly external to the mid-Poupart line. Upon the lateral wall of the abdomen tenderness is elicited upon pressure exerted at the same level. Posteriorly renal tenderness is elicited upon exerting pressure in the costovertebral angle.

The *form* of the kidney, even in pronounced enlargements of the organ is usually maintained to such a degree that the organ may be recognized by its reniform contour. Frequently it is considerably elongated; and in the case of the polycystic kidney is studded with regularly spherical nodules.

The *volume* of the kidney in the presence of pathologic changes of the organ presents all gradations from the very moderate enlargement of the gland which scarcely renders it palpable to immense hypertrophy which fills a considerable portion of the abdominal cavity.

The examiner not infrequently encounters difficulty in the case of the pathologic kidney in establishing its limits in respect to the neighboring abdominal organs. The inflamed kidney or the kidney which is the seat of malignant disease is very prone to contract adhesions with adjacent viscera, particularly with the liver and with the spleen. Occasionally it is possible to palpate the hilum of the organ and to appreciate the pulsation of the renal vessels in this situation.

The range of *mobility* of renal enlargements is readily studied in the case of moderate hypertrophies of the organ, and with considerable difficulty in the case of excessive enlargement of the kidney. In the moderately hypertrophied kidney it is readily determined upon bimanual palpation that its range of mobility is backward into the loin, while it possesses very little or no lateral mobility. This radius of mobility of the kidney is of considerable aid in differentiating renal enlargements from a distended gall bladder, which presents a distinct lateral mobility. Similarly, the kidney possesses no true respiratory mobility in contradistinction from hepatic growths which possess this mobility to a striking degree.

The *state of the surface* of the diseased kidney is variable. Renal hypertrophies with smooth surfaces point to hydronephrosis and cystic disease of the organ; while those with irregular, nodular surfaces point to solid tumors of the organ, notably to carcinoma and sarcoma, and to the polycystic kidney. In cases of renal hypertrophy in which the colon is carried forward in front of the organ and is compressed between its anterior face and the abdominal wall the intestine is occasionally palpable in the form of a rounded band crossing the anterior surface of the kidney.

The *consistency* of the kidney in the presence of renal hypertrophy is frequently of considerable aid in the determination of the nature of the underlying lesion. In the case of solid tumors of the organ the tumor is hard and unyielding upon palpation. In the case of hydronephrosis, on the contrary, the organ is soft and yielding, and fluctuation is occasionally to be detected. Fluctuation is elicited most readily when the walls of the organ are not unduly tense; and an increase in the quantity of the fluid and of the tension of the walls renders the fluctuation less distinct. In the presence of echinococcus disease of the kidney it is occasionally possible to elicit hydatid fremitus over the site of the cyst.

Occasionally the examiner may be confused by a solid mass which crosses the median line low down in the abdominal cavity and which is continuous with the inferior poles of the kidneys. In this connection he should think of the possibility of the existence of the congenital fusion of the inferior poles of the organ with the production of the "horseshoe kidney," which may be situated as low as the concavity of the sacrum.

Occasionally a horseshoe deformity of the kidneys has imparted to it the systolic pulsations of the subjacent aorta, simulating with its rise and fall an aneurysm of this vessel.

Moreover, upon palpation of the renal region, the examiner may encounter a sensation of diffuse resistence to the palpating hand rather than a distinct tumor. In this event he should think of the possibility of purulent infiltration of the perirenal tissues or perinephritic abscess and should carefully palpate the costovertebral angle and endeavor to elicit fluctuation if such be demonstrable.

In the case of the left kidney, owing to the fact that there is no anatomic attachment of the kidney to the descending colon, the colon is frequently displaced, allowing the renal tumor to come forward and meet the abdominal wall. However, in these circumstances, the displaced colon will yield a tympanitic note along the border of the solid mass. Tumors of the spleen reach the abdominal wall above the transverse colon, which is displaced downward.

After palpating the left kidney region the hand is pressed downward along the left side of the abdomen, palpating as deeply as possible but always flexing the fingers from the metacarpophalangeal joint with the fingers themselves straight. Clawing or probing movements produce contraction of abdominal muscles as already noted.

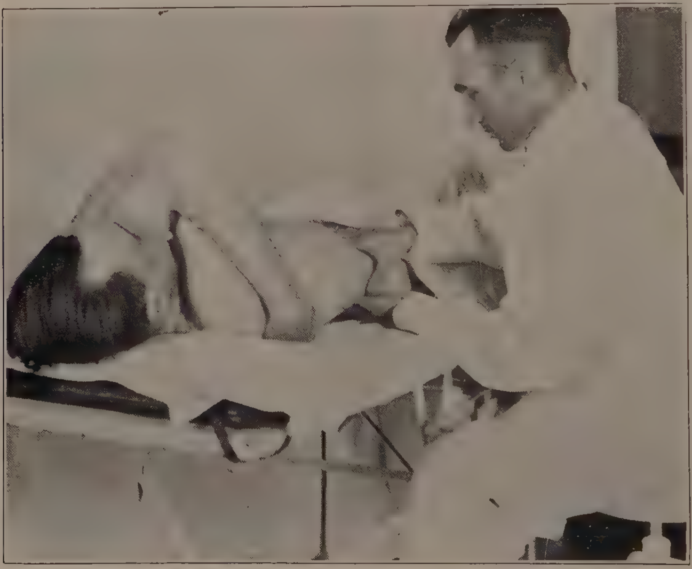


Fig. 152.—Palpation of the right kidney.

Frequently a cord-like mass may be felt. This is the descending colon which may be more or less tender in the normal subject. Fecal masses in the colon may give rise to doughy feeling tumors which may be moved along the colon. The right hand is then moved across the abdomen to the right kidney region which is palpated by placing the left hand over the lumbar muscles at the level of the ninth and tenth ribs and the right hand on the abdominal wall at a point directly opposite. The subject is instructed to take a deep breath and at the end of inspiration the left hand is pushed upward and the right downward. The lower pole may be felt to slip under the fingers of the right hand at the beginning of expiration.

When a distinct growth is palpated in the right renal region doubt may arise as to whether or not the growth is of renal origin or whether it is an enlarged gall bladder, or whether it springs from the liver, spleen, pancreas, or the pylorus. In this connection the examiner should recall that a tumor of the kidney is situated more laterally than is a distended gall bladder, and exhibits its principal radius of mobility backward into the loin, while that of the gall bladder is manifested transversely below the inferior margin of the liver. Also, as stated in a previous paragraph, the ascending colon is attached by areolar tissue to the inferior pole of the right kidney so that a renal hypertrophy carries the tympanitic colon before it, while tumors of the gall bladder, pylorus, or pancreas reach the abdominal wall nearer the median line, without the intervention of the tympanitic colon.

Hepatic enlargements exhibit distinct respiratory mobility, which is absent in the case of renal tumors; hepatic enlargements develop above the transverse colon, which they displace downward; and, as Bright showed, in the case of renal enlargements it is possible to insinuate the palpating hand between the growth and the anterior abdominal wall, while in the case of hepatic growths this maneuver is impossible.

THE LIVER

In practicing palpation of the inferior hepatic border the subject should assume the dorsal decubitus with the knees drawn up and supported. The examiner, upon the right side of the patient, should place the right hand upon the abdominal surface immediately below the right costal arch, and by gradually sinking the fingers into the abdominal wall, should seek for the lower margin of the liver. If it be found that the liver projects below the costal margin, the examiner should apply both hands flat upon the abdomen, and by downward pressure cause the finger tips to glide over the exposed portion of the liver, searching for tenderness, any abnormality in contour, and investigating the consistency of the organ.

In the normal adult subject the inferior border of the liver will not be encountered below the costal arch in the male subject. In the female subject, on the contrary, who has worn clothing which constricts the lower portion of the thorax, the inferior border of the liver occasionally occupies a position below the right costal margin. In multiparæ the relaxation of the abdominal walls and of the suspensory ligaments of the liver tends further to produce lowering of the liver in these subjects. In young children the inferior border of the liver is very frequently encountered below the costal margin.

If the inferior margin of the liver is not encountered below the costal margin, the examiner should place both hands upon the abdominal surface just below the costal arch and should press inward and upward beneath the costal arch as far as is possible. The subject is directed to inspire deeply, when during inspiration the liver will be felt to descend with the descent of the diaphragm and to become palpable at the completion of inspiration. By the procedure first described an enlarged liver or a depressed liver will be revealed projecting below the costal arch; while, by the second maneuver, a normal or contracted liver may frequently be palpated.

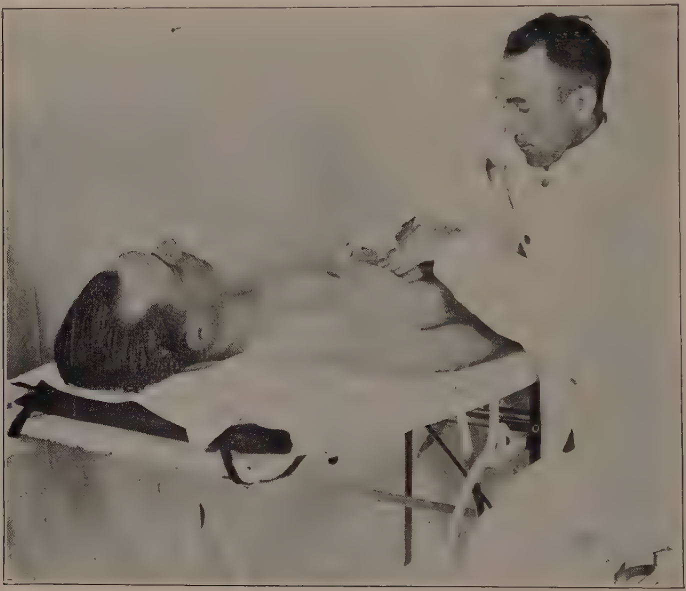


Fig. 153.—Palpation of liver.

The facility with which the liver is palpated is markedly influenced by the state of the abdominal walls. An excessive deposition of fat in the walls impedes and occasionally renders quite unsatisfactory the exploration of the liver by palpation. In the multipara, on the contrary, with relaxation of the abdominal walls and with possible diastasis of the recti, the manual exploration of the organ is rendered so much the more easy.

Similarly, the presence of gas or fluid in the intestinal tract or in the abdominal cavity is a frequent cause of confusion in palpation of the liver. In the presence of ascites, which is so often due to hepatic affections and renders a careful palpation of the organ so desirable, the examiner is occasionally able to reach the inferior border of the liver by exercising a few rather sharp, jerky palpating movements toward the lower border of the organ, which by crowding the fluid aside enables him to reach the liver. In this class of patients the examination is further facilitated by placing the patient in the left lateral decubitus or in the genupectoral position. In the case of ascites of suspected hepatic origin, the fluid should be evacuated from the abdominal cavity, whereupon palpation of the liver becomes very easy and is apt to reveal some very striking findings.

Enlargement of the liver accompanies the acute infectious fevers, fatty infiltration of the organ, chronic passive congestion of the organ, and amyloid disease of the liver. Similarly, hepatic enlargement attends Weil's disease, hepatic abscess, carcinoma or gumma of the organ, leukemia, hypertrophic hepatic cirrhosis, echinococcus cyst, and Banti's disease.

In every case in which enlargement of the liver is suggested by the finding of a palpable inferior margin of the liver, the examiner should continue the examination in the effort to determine the state of the surface of the liver, the consistency of the organ, the presence of sensibility of the liver, and the respiratory mobility of the mass.

When the inferior border of the liver is appreciated by the palpating hand, it may be found to be smooth or to be irregular and the site of nodular elevations and depressions. When a smooth surface is encountered, the examiner should proceed to identify it with the liver by establishing upon its surface the two notches of the inferior hepatic border, the one approximately one inch to the right of the median line and the second at the outer border of the right rectus muscle.

An irregular, nodular hepatic surface is encountered in carcinomatous infiltration of the liver, in atrophy of the organ of syphilitic or nonspecific origin, and in echinococcus disease of the organ. The nodules of carcinoma present a central umbilication, which can occasionally be appreciated in palpation of the liver through a thin abdominal wall. The nodules of the syphilitic liver are uniformly small, whereas the irregularity of the liver in the case of echinococcus disease takes the form of a few protrusions of relatively extensive dimensions.

The palpable *consistence* of the liver occasionally gives a clue to the nature of the cause of the enlargement. The fatty liver possesses a consistance scarcely altered from that of the normal organ, while that of chronic passive congestion scarcely offers any more resistance. In the case of amyloid disease of the liver, on the contrary, the organ frequently attains a woody hardness. Carcinomatous infiltration of the liver produces a liver of hard consistence. In the case of hepatic abscess and echinococcus cyst the consistence of the mass is softened and fluctuation is frequently obtained under these circumstances. Frerichs has found echinococcus disease of the liver, however, associated with the development of multilocular cysts of the organ, in which the liver possessed a consistence almost cartilaginous, suggesting strongly carcinomatous infiltration of the organ. Jaccoud has encountered fluctuation of the liver in certain cases of hypertrophic cirrhosis of the organ.

Sensibility of the liver, manifested by pain upon pressure, may be diffuse or may be circumscribed to certain definite regions of the abdomen. Diffuse sensibility of the liver is encountered in fatty degeneration and chronic passive congestion of the organ, in hypertrophic cirrhosis, in hepatic abscess and carcinoma, and in inflammatory disease involving the peritoneal covering of the organ in perihepatitis.

The respiratory *mobility* of the inferior border of the liver, which was described under inspection, can be readily detected upon palpation of the inferior border of the organ. During inspiration this border of the organ descends, only to ascend to a corresponding degree during expiration. The respiratory mobility of masses of hepatic origin is a valuable sign in differentiation from similar masses springing from the stomach, intestine, kidney, or pancreas.

But while the growths springing from the liver and from the spleen alone among intraabdominal growths possess a true respiratory mobility, it does not follow that all hepatic growths are mobile. The presence of extreme tympanites or ascites impairs or prevents the respiratory mobility of certain hepatic enlargements. Similarly, when an enlarged liver has established firm adhesions with the abdominal walls, respiratory mobility of the organ is abolished. Also as inflammatory disease of the liver is very frequently accompanied by inflammation of its peritoneal investment, the patient in the presence of this affection frequently volitionally inhibits the respirations, with consequent abolition of the respiratory mobility of the liver. In the presence of extreme enlargement of the liver, moreover, the prolonged pressure of the enlarged organ upon the right vault of the diaphragm is capable in certain instances of provoking atrophy of this muscle, with consequent abolition of respiratory mobility of the liver.

Occasionally one can appreciate a tactile fremitus upon palpation of the abdominal wall over a liver which is the seat of perihepatitis; but this finding is very inconstant, and it is more common to detect friction of the roughened peritoneal surfaces upon auscultation than it is to detect the fremitus during palpation.

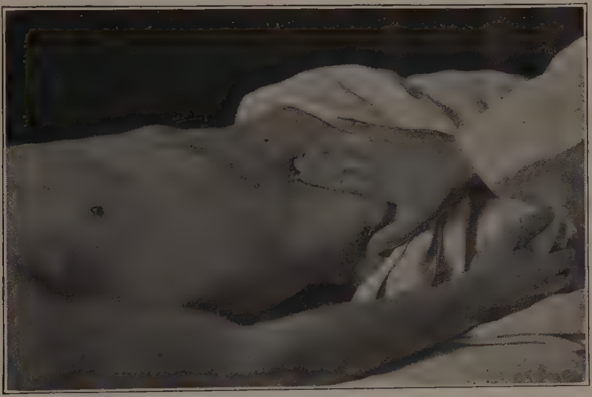


Fig. 154.—Palpating for tenderness in right lower quadrant. In first step three fingers are applied lightly to integument.

Disease and tumors of the gall bladder deserve particular attention during palpation of the upper abdominal quadrant. Numerous technics have been elaborated for the detection of the tenderness of gall bladder disease. Simple manual palpation of the abdomen may reveal tenderness at *Mayo Robson's point*, which corresponds to the junction of the outer and middle third of a line drawn from the ninth costal cartilage to the umbilicus.

RIGHT LOWER QUADRANT

After palpating the liver region one palpates the right lower quadrant. First palpate with the hand lightly placed over the appendix region, the central point of which is the middle of a line drawn from the umbilicus to the crest of the ilium. Should no tenderness appear on light palpation the hand should be applied more firmly and finally three fingers reinforced by the left hand may be sunk deeply into the abdominal wall at this point. By extending the leg on the thigh and flexing the thigh on the abdomen the psoas muscle is contracted, forcing the structures overlying this muscle upward. This procedure may sometimes render the appendix palpable or increase the tenderness (Meltzer's sign).

Palpation of the lower abdomen just above the middle of Poupart's ligament may elicit tenderness when ovaries are enlarged or inflamed. Palpation over the suprapubic region with the flat hand gradually working deep under the pubic bone is used to elicit tenderness or tumors of the bladder. (A distended bladder may produce a rounded mass rising above the pubes, sometimes almost to the umbilicus.)

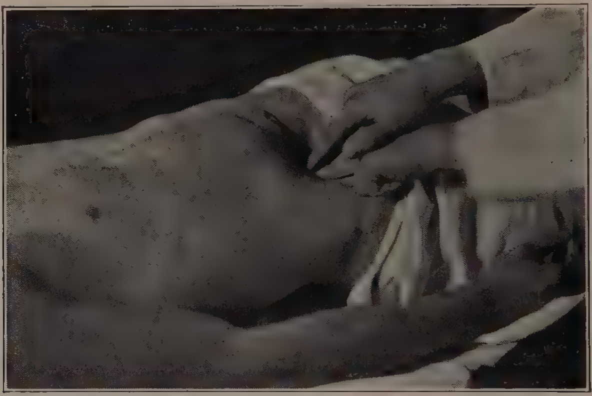


Fig. 155.—Palpating for tenderness in right lower quadrant. In second step palpating fingers are sunk deeply into wall, reinforced by other hand.

THE UMBILICUS

Palpation of the umbilicus, using the index or index and second fingers, noting any rigidity of the umbilical region, the subject should always be instructed to cough while the fingers are over the umbilical region, as weakness of the umbilicus or hernia will produce a distinct impulse on coughing.

THE SMALL INTESTINE

The soft yielding walls of the normal small intestine are not to be felt upon palpation of the abdominal wall. But in the subject with thin abdominal walls enteroliths or large gallstones may occasionally be detected by palpation if they are present.

A hard tumor which is encountered in the neighborhood of the umbilicus and in the lower umbilical and upper hypogastric regions is very apt to belong to the small intestine, and may be

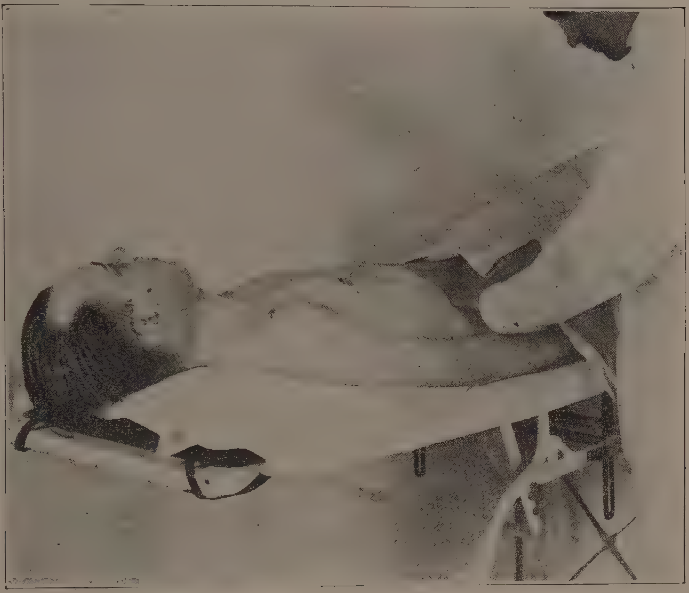


Fig. 156.—Palpation of ascending colon.

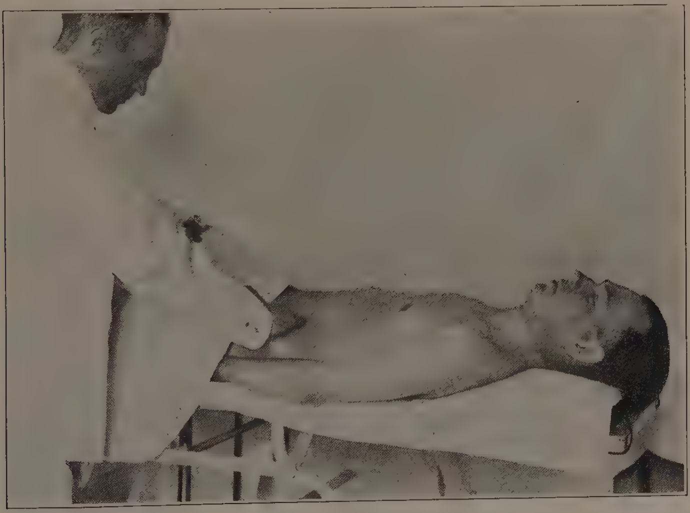


Fig. 157.—Palpation of the descending colon.

significant of malignant disease, of intussusception, or of volvulus. A solid mass encountered in these regions, however, is occasionally due to matting of the omentum in tuberculous peritonitis, constituting tabes mesenterica.

When an intestinal tumor pulsates, the patient should be placed in the genupectoral position, in order to determine whether the pulsation is transmitted to the tumor from the subjacent abdominal aorta. When palpation of the intestine is practiced in this posture, the tumor falls away from the aorta, and the pulsation of this vessel is no longer imparted to the growth.

THE COLON

In the palpation of a tube of such extensive distribution and one which possesses such a variety of anatomical relations in different portions of its distribution, it is evident that a maneuver which is applicable to one portion of the tube is not applicable in other portions. As a result of the changes in direction of the colon with reference to the different diameters of the abdominal cavity, it is necessary to palpate different portions of the tube in different planes.

In palpation of the *ascending colon* the patient should in the first instance assume the dorsal decubitus with the knees drawn up and supported; and, if examination in this posture is not satisfactory, the patient should be placed in the left lateral decubitus in order to cause the coils of the small intestine to fall away toward the left side of the abdominal cavity, leaving the fixed ascending colon free to manual palpation.

With the patient in the dorsal decubitus the examiner assumes a position by the right side of the patient and applies the finger-tips to the abdominal wall along the distribution of this portion of the intestine at right angles to the course of the ascending colon. By sinking the finger tips into the abdominal wall and moving them outward with a sinking, rolling movement, the ascending colon frequently is felt between the finger tips and the posterior abdominal wall, and the examiner may determine whether it is distended or contracted, whether its wall is free or is studded with nodulations, and whether it contains fecal masses. In this connection, it is well to remember that fecal accumulations are more frequently encountered in the cecum and ascending colon than elsewhere.

If the palpation of the ascending colon in the dorsal decubitus is not entirely satisfactory, the maneuver should be repeated with the patient occupying the left lateral decubitus with the knees drawn up in order to relax the tension of the abdominal muscles.

As the ascending colon is moored to the anterior face of the right kidney by areolar tissue, this portion of the intestinal tract is freely movable with the kidney; and in the presence of renal enlargement the ascending colon is pushed forward in front of the enlarged kidney.

During palpation of the *transverse colon* the examiner places both hands flat upon the abdomen in the umbilical region with the finger tips extending somewhat above the level of the surface markings of the normal colon as it dips into and ascends from this region of the abdomen. The patient is directed to inspire deeply; at the commencement of the following expiration the examiner makes downward pressure with the finger tips and endeavors to determine the position and state of this portion of the intestine. In palpation of this section of the colon, as in the case of other portions, the hand should be applied to the abdominal surface at right angles to the course of the colon. Hence, if the transverse colon pursues an arched course with the convexity directed downward, as is very frequently the case, the right and left halves of this portion of the intestine must be palpated in different directions. While the normal transverse colon is encountered at the level of or above the umbilicus, in enteroptosis it may be encountered as low as the symphysis pubis.

THE PANCREAS

The examiner will very rarely be able to palpate the normal pancreas, save possibly in the emaciated subject with diastasis of the rectus muscles. Leube and Ewald have been able to palpate the normal organ when the stomach and intestine were empty; but as a rule, the examiner may anticipate considerable difficulty in palpation of the normal pancreas.

When a mass is encountered in the deeper portion of the lower epigastric or upper umbilical region, it may be due to a solid tumor or a cyst of the pancreas; and pancreatic solid tumors in the vast majority of cases are carcinomatous.

A tumor arising from the pancreas is apt to be mistaken for a tumor of the pylorus, a distended gall bladder, aortic aneurysm, or a tumor of the transverse colon. A pyloric tumor is always more superficial than is a pancreatic growth. Moreover, a tumor at the pylorus is freely movable, in marked contrast to the immobility of pancreatic tumors, and pyloric tumors are prone to produce pyloric stenosis with consequent gastrectasis. A distended gall bladder exhibits lateral mobility; it is more superficial than is a tumor of the pancreas; and it is not accompanied by glycosuria and fatty stools as are pancreatic growths very frequently.

A pancreatic tumor may be elevated with each pulsation of the abdominal aorta, simulating aortic aneurysm; but while the pulsation of aortic aneurysm is expansile and is exercised in all directions, that of a superjacent pancreatic tumor is not expansile, the mass being simply elevated with each pulsation of the subjacent aorta.

THE EPIGASTRIC REGION

The epigastric region is usually the last portion of the abdomen to be palpated, as the underlying solar plexus makes it tender even in the normal subject. The palms of the hands should lie flat upon the epigastrium immediately below the tip of the ensiform cartilage and gradually sink them into the abdominal wall, covering this area progressively in a direction from above downward. Following this maneuver, he should apply the tips of the fingers of the right hand to the lower portion of the epigastrium; and, with the fingers well separated, search for the greater curvature of the stomach by a series of gentle, pushing movements.

Palpation of the epigastrium may reveal the presence of tenderness, may confirm visible peristalsis, or the presence of a tumor of the stomach, or may elicit succussion sounds.

Excessive tenderness elicited in the epigastric region, in which such a large number of vital structures lie within a limited space, naturally possesses a varied significance. It may be significant of simple inflammation of the mucous lining of the stomach, of ulcer or carcinoma of the stomach, of pancreatic carcinoma or carcinoma of the common bile duct, or of acute hepatis, hepatic colic, pancreatic calculus, or acute pancreatitis.

In the case of gastric ulcer the tenderness is circumscribed in the majority of cases to a point corresponding to the junction of a vertical line drawn from the umbilicus to the tip of the ensiform cartilage, and of a horizontal line connecting the free margins of the eighth costal cartilages. Moreover, gastric ulcer is commonly attended by a painful pressure point situated immediately to the left of the body of the twelfth dorsal vertebra.

The tenderness of acute gastritis and of gastric carcinoma are diffuse, pervading all portions of the epigastrium upon deep pressure. Occasionally, also, in the case of gastric carcinoma it is possible, in patients with thin abdominal walls or with diastasis of the recti, to palpate the carcinomatous nodules upon the anterior gastric wall.

A solid tumor of the stomach may occupy the anterior wall, the posterior wall, or the pylorus. A pyloric tumor is commonly palpable in a circumscribed area to the right of the umbilicus and a little above this level. Moreover, pyloric tumors are commonly attended by varying grades of pyloric stenosis with visible peristalsis and an abnormally low position of the greater curvature of the stomach. The stomach in this state commonly yields distinct succussion sounds even several hours after the ingestion of food or fluid.

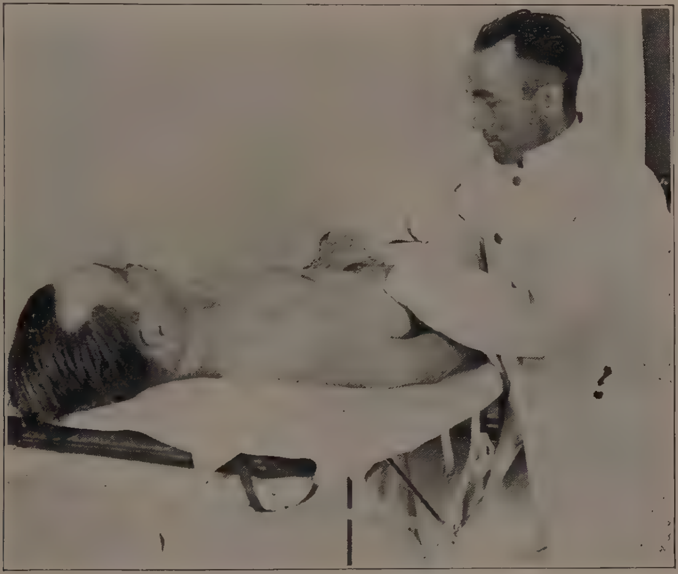


Fig. 158.—Palpation of the epigastrium.

A solid tumor of the anterior gastric wall is readily palpable by ordinary methods; but in the uninflated stomach it is impossible to say whether a palpable tumor is situated upon the anterior or the posterior wall of the viscus. In the determination of the site of the tumor under these circumstances artificial inflation of the stomach with carbon dioxide is very serviceable. When so distended, the tumor situated upon the posterior wall is no longer freely palpable.

The soft, yielding walls of the normal stomach are not palpable except in the cases in which the organ has been artificially inflated. In the artificially inflated stomach the examiner feels upon palpation of the epigastrium a sensation of resistance when the stomach is reached, a sensation closely akin to that which is experienced upon palpating a rubber bag filled with gas. In the presence of gastrectasis and gastroptosis the greater curvature of the organ is encountered at an abnormally low level; and in the case of gastroptosis it is frequently possible in the inflated organ by palpation to outline the lesser curvature of the organ and to establish the fact that the superior limit of the stomach has departed from its normal habitat beneath the left lobe of the liver.

Succussion sounds arising within the stomach are elicited by palpating the epigastrium with a series of short pushing movements with the finger tips, beginning well below the normal site of the greater curvature and proceeding upward until this point is reached, whereupon the splashing sounds will become audible. It is not sufficient, however, to cease the palpation when the sounds become audible, unless at the same time the fluid becomes palpable.

Gastric splashing sounds were first described by Chomel, who regarded them as an invaluable sign of gastrectasis; and so they are if they fulfill certain topographical and chronological conditions. These conditions have been established by Bouchard; namely, that splashing sounds are indicative of gastrectasis when they extend well beyond the limits occupied by the normal organ; and when they are elicited at least six hours after the ingestion of food or fluid.

SPECIAL METHODS OF PALPATION

After completing routine palpation of the abdomen, the gall bladder region may be palpated by placing the ball of the thumb just below the costal margin in the right epigastric region and forcing it deeply under the ribs at the end of inspiration. This may be done over several points in the region of the gall bladder. If tenderness is noted similar pressure with the thumb at a corresponding location in the left side is useful.

Palpation of the kidneys with the patient in the erect position is best accomplished by placing the left hand on the lumbar muscles and the right just below the ribs. At the end of a deep inspiration an attempt to bring the hands together by firm pressure is made. The lower pole of the right kidney can sometimes be grasped and felt to slip beneath the fingers on expression.

Bimanual palpation of the liver with the hands similarly placed over the lower ribs is used, especially to elicit liver pulsation.

Palpation of the abdominal wall with the subject in the knee chest position may bring the intestines against the abdominal wall and render certain movable tumors more easily palpable.

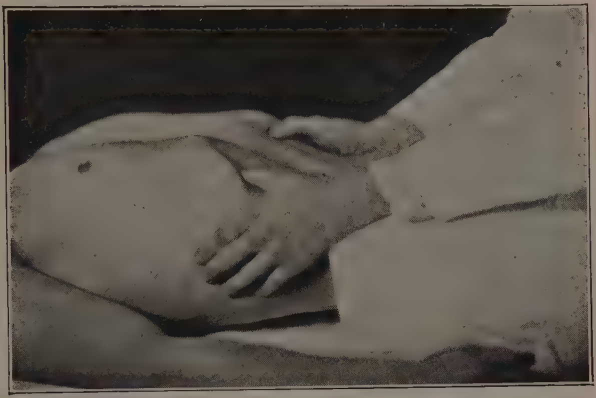


Fig. 159.—Thumb palpation of the gall bladder.

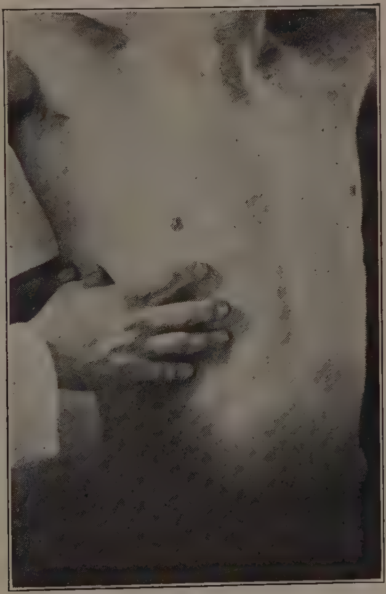


Fig. 160.—Bimanual palpation of the liver.

Palpation of the abdomen with the subject in a hot bath or under partial or complete anesthesia is occasionally useful when the abdomen is very rigid or painful.

THE ABDOMINAL WALL

An estimate of the thickness of the abdominal wall may be made by pinching up the wall between the forefinger and thumb, or by approximating the two hands placed palm downward upon the abdominal surface. Increased thickness of the abdominal wall indicates an excess of fat, the presence of edema, or suppuration of the wall. If the increase be due to excessive deposition of fat in the wall, a fat wave will be obtained upon bimanual palpation; if due to edema, the wall will pit upon pressure; and if due to a localized or extensive suppuration of the wall, there will be accompanying signs of inflammation, as discoloration of the surface, and elevated temperature.

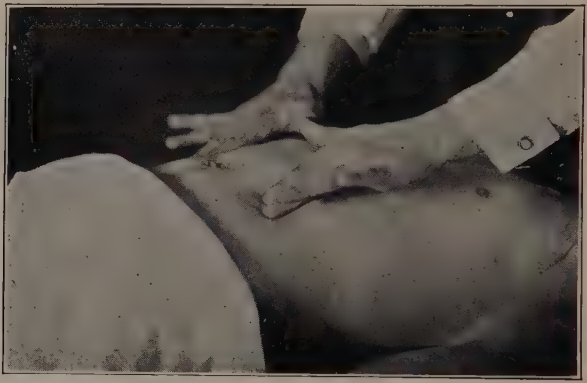


Fig. 161.—Testing thickness of abdominal wall. Fingers applied lightly to abdominal surface.

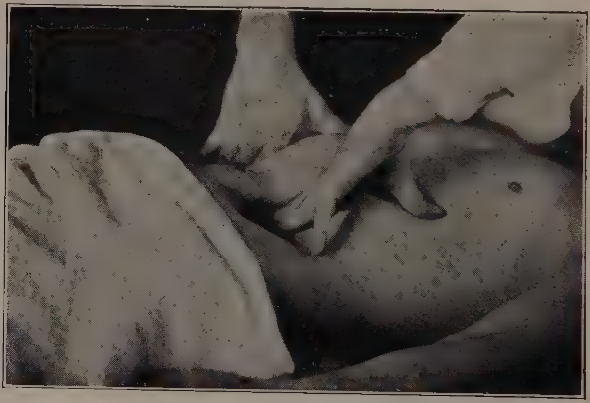


Fig. 162.—Testing thickness of abdominal wall. Fingers carried beneath the wall.

Rigidity of the abdominal wall with possibly spasm upon attempts at palpation, indicates inflammation of the peritoneum or of an abdominal organ. Muscular rigidity is most commonly encountered in the rectus muscle. Rigidity of the right rectus alone occurs with acute appendicitis, whereas bilateral rigidity of the recti accompanies acute peritonitis.

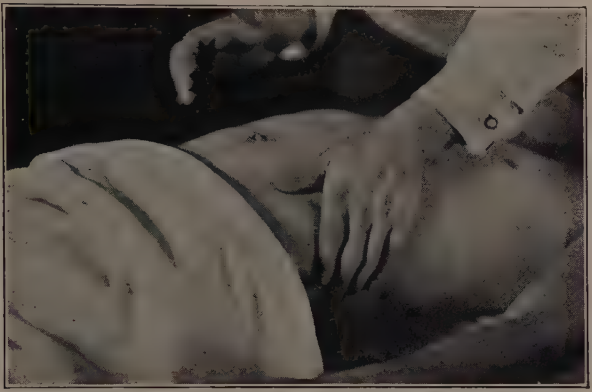


Fig. 163.—Testing for a fluid wave across the abdomen.

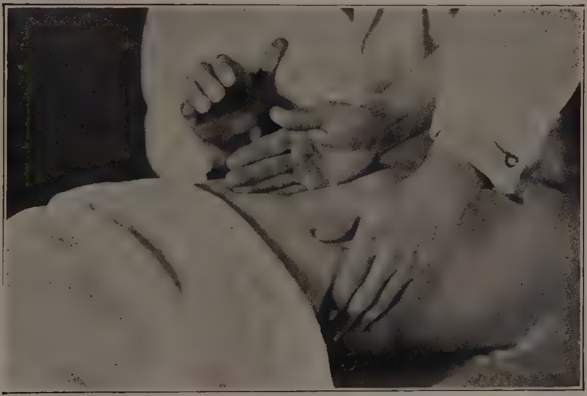


Fig. 164.—Differentiating a fat wave from a fluid wave. The fat wave is stopped by the pressure in the median line.

Tenderness.—When tenderness is elicited upon palpation of the surface of the abdomen, if not due to hyperesthesia of the parietes, it points to a diseased abdominal organ. The tenderness is most apt to be encountered over the gall bladder, stomach, spleen, kidney, appendix, and sigmoid flexure. In acute peritonitis there is general or diffuse tenderness.

Fluid Wave.—In the presence of ascites a fluid wave can be demonstrated upon bimanual palpation. In palpating for a fluid wave in the peritoneal cavity, one hand of the examiner is applied flatly over one lumbar region, while the opposite side of the abdomen is tapped with the finger tips of the other hand of the examiner. An impulse or wave is thus created in the fluid, which is appreciable to the palpating hand.

Fat Wave.—Excessive deposition of fat in the abdominal wall gives a wave upon bimanual palpation closely simulating the fluid wave. To exclude such a fat wave during bimanual palpation an assistant should apply the ulnar side of the hand to the median line of the abdomen while the examiner practices bimanual palpation as in eliciting the fluid wave, when the fat wave is interrupted by the intervention of the assistant's hand and is not transmitted to the palpating hand of the examiner.

Intraabdominal Tumor.—An intraabdominal tumor may be so large as to entirely fill the abdominal cavity; but as a rule careful bimanual palpation enables the examiner to determine its origin, its size and shape, whether it is fixed or movable, and finally whether it moves with respiration. In examining for respiratory mobility of an intraabdominal tumor the examiner should place both hands palms downward flat upon the abdomen, with the fingers directed toward the costal arch, while the patient is directed to breathe deeply. At the commencement of expiration, the finger tips are pressed downward firmly and with uniform pressure, when the lower margin of the tumor or enlarged organ is encountered. The most commonly encountered movable abdominal tumor is a movable or displaced kidney; but tumors of the liver and spleen are movable with respiration.

Peritoneal Friction Fremitus.—Upon palpating over the upper regions of the abdomen a friction fremitus analagous to pleural or pericardial friction fremitus is sometimes encountered. The vibration is produced by roughening of the peritoneal surfaces in peritonitis. This fremitus is most commonly encountered over the hypochondriac regions, in the presence of peritoneal involvement in the course of perihepatitis or perisplenitis.

CHAPTER XX

PALPATION OF THE INGUINAL, GENITAL, AND PERINEAL REGIONS

THE INGUINAL REGION

Inguinal glands are palpable in many individuals who give no history of venereal or other infection. The inguinal chain is just below and parallel to Poupart's ligament. The glands are usually discrete, but following genital infections masses of glands are matted together, forming tumors of considerable size.

Enlarged inguinal glands may result from syphilis, gonorrhea, or chancroid, or may result from infection of the foot, leg or thigh. Lymphatic leucemia also produces enlargement of the inguinal glands.

Scars on the inguinal region may be superficial, or may involve the deeper structures sufficiently to cause obstruction to the venous return, thus producing edema of the lower extremity.

Tumor masses in the inguinal region other than enlarged glands may be due to femoral or inguinal hernia. When a tumor mass shows expansile pulsation when the subject increases intraabdominal tension, as by coughing, that tumor is either a hernia or connected with a hernia.

Abscesses in the inguinal region frequently fluctuate and this fluctuation should be noted in connection with impulse on coughing, as abscesses have been confused with hernia and may occur with it.

THE GENITALS

Male

All scars about the penis should be palpated for tenderness and fixation to deep structures. Eruptions and ulcers should also be palpated for induration.

Chancre is a round indurated elevation, frequently ulcerated.

Chancroid is usually irregular with indurated edges and a crater-like center.

The scrotal veins are frequently enlarged and tortuous, giving the feel of a mass of worms (varicocele). The testicles should be compared both as to the body of the testicle and the epididymis. The epididymis is often enlarged and painful in acute gonorrhea and occasionally from nonspecific inflammation or malignancy.

Malignant tumors of the testicle are common in elderly men and appear as hard nodular enlargements, more or less tender. Hydrocele may distend the scrotum to enormous size. This tumor has a peculiar resilient feel like a rubber ball filled with water.

Atrophy of one or both testicles may occur. It is most common following mumps. One or both testicles may remain in the inguinal canal, the so-called undescended testicle. Absence of a testicle may result from surgical removal or atrophy.

After palpating the contents of the scrotum the index finger should invaginate the scrotum to reach both right and left inguinal rings. On reaching the external inguinal ring the subject should be instructed to cough. Weakness of the ring will cause distinct impulse and if hernia is present a portion of the small intestine may be forced through the ring.

Female

The external labia should be palpated between the thumb and finger for scars or tumor masses. When the subject is instructed to cough an inguinal hernia may present in the external labium.

Internal palpation is best accomplished with the patient lying on a firm table with the knees flexed on the thighs, and the thighs flexed on the abdomen. The knees should be widely separated. The gloved forefinger of the left hand is inserted into the vagina while the right is placed on the abdominal wall just above the pubes. The urethral orifice and clitoris should first be palpated for enlargement and tenderness. The palpating finger is then swept around the interior of the vagina, noting areas of tenderness or enlargement. It is then pressed downward and backward to encounter the cervix. The size, consistency and sensitiveness of the cervix should be noted, also the size and shape of the os. In women who have borne children the cervix is frequently enlarged and the os irregular and patulous. A firm nodular cervix should suggest malignancy, while a soft cervical tip in the presence of other signs and symptoms would suggest pregnancy. After palpating the cervix the right hand is pressed firmly downward in an attempt to feel the body of the uterus. If normally placed, in women before the menopause, it appears as a firm mass between the palpating finger and the right hand. In women who have passed the menopause it may not be palpable. The diseased or pregnant uterus may vary greatly in size and consistency. For details of changes in size and form of the uterus, works on gynecology and obstetrics should be consulted.

By passing the finger to the right and left of the cervix and moving the right hand on the abdominal wall to the right and left of the midline to make abdominal pressure, one may discover enlarged and tender ovaries or fallopian tubes. Details of such enlargements will be found in works on gynecology.

The pelvic floor is then palpated for evidence of rupture or weakness of the perineal muscles. When considerable weakness of the pelvic floor occurs the rectum or bladder may protrude beyond the vaginal orifice (rectocele or cystocele). Increasing intraabdominal pressure by straining or coughing will accentuate such protrusions.

THE PERINEAL REGION AND RECTUM

The external perineum is palpated for weakness, scars, and tumors. Abscesses of the perineal region are common and appear as exquisitely tender tumors, often fluctuating. The gloved finger is then inserted into the rectum, noting the feel as it passes the external and internal sphincter.

In the male, the prostate should be felt as a bilateral mass in the anterior surface of the rectum. It is firm and normally only moderately tender. Other masses within the rectum may be due to dilated hemorrhoidal veins which, when thrombosed, are hard and often very tender. Malignant growths in the rectum may be felt as hard, tender, nodular masses.

In the female, the uterus can frequently be felt beneath the anterior rectal wall, somewhat higher than the region in which the prostate is felt. The uterus is an elongated mass not lobulated, and not as firm as the prostate. It is frequently freely movable, although following pelvic inflammation it may be fixed.

With the left hand on the abdominal wall the rectal method of palpation of the uterus is often the only method available in examining unmarried women.

CHAPTER XXI

PALPATION OF THE LOWER EXTREMITIES

THE FOOT

The toes should be palpated much as are the fingers, for joint movements, tumors, and painful calluses. The first tarso metatarsal joint is subject to painful and deforming enlargements in gout and "bunions."

The plantar arches should be palpated for evidence of weakened muscles or foot strain. The ankle joint movements should be compared as to freedom of motion, pain, and enlargement. The region below the external malleolus is the first to show evidence of edema due to decompensated heart or kidney, or in venous obstruction. Attempts to pit the tissues in this region with the forefinger should be part of the routine of palpation.

The dorsalis pedis artery is frequently partially or wholly obliterated from arteriosclerosis or obliterating endoarteritis. Its pulsation should be carefully felt and right and left should be compared.

THE LEG

The veins on the leg often show hardened areas from thrombosis. The "feel" of enlarged veins should always be noted.

Musculature of legs should be compared and the strength tested in various movements. Paralysis of the extensor muscles occurs from ingestion of toxic amounts of arsenic, alcohol and lead. Anterior poliomyelitis may leave various groups of muscles paralyzed or weak.

The patella should move freely from side to side and should be smooth in contour. Fracture of the patella often leaves the knee permanently stiffened. Arthritis, fractures, and dislocations may also impair the movement of the knee joint. Enlargements of the knee may be due to fluid in the joint capsule or enlargement of the joint structures. Arthritis, due to acute rheumatism, tuberculosis or syphilis (Charcot's joint), are the most common causes.

THE THIGHS

The musculature of the thighs should be compared as to tonicity. Variations between the right and left are common in normal individuals. The upper portion of the thighs should be palpated with the finger tips for enlarged glands and tender veins. Tumors of the thigh may be fatty or due to sarcoma of the femur.

Movements and contour of the hip joints should be carefully noted. Dislocations and fractures often leave some restriction in motion of the hip joint and more or less palpable deformity.

Abscesses of the gluteal region are often of considerable size. They may be due to pyogenic or tubercular infections. They should be tested for fluctuation.

X-ray examinations are indispensable aids to the diagnosis of bone and joint diseases.

SECTION V

PERCUSSION

CHAPTER XXII

OBJECT AND TECHNIC OF PERCUSSION

Percussion was first applied to the exploration of pathologic states of the respiratory organs by Auenbrugger of Vienna in 1761. However, the value of percussion was not fully appreciated until the year 1808, when the French physician Corvisart, physician to Napoleon I., revived the subject and further elaborated the technic of percussion. During the same epoch Piorry of France introduced topographical percussion of the thoracic viscera, and Skoda of Vienna published his studies upon the probable genesis of hyperresonance and tympany in connection with pulmonary relaxation and compression. The technic of percussion and the attributes and mode of production of the various percussion findings were further elucidated by the epoch-making researches of such investigators as Wintrich, Traube, Biermer, Gerhardt, and Weil. Indeed, so thorough were the studies undertaken by these latter investigators, that modern investigation has accomplished little in the way of additions to the physical laws which were enunciated by these authors in relation to the generation of the various characteristics of the percussion sounds.

Percussion is employed in the study of disease of the respiratory organs for the purpose of eliciting sounds which are normal to the pulmonary parenchyma and sounds which only arise in the presence of diseased states of these organs; to determine the situation of the borders of the intrathoracic organs; and also to note the degree of resistance offered to the percussion stroke by the tissues under examination.

Percussion, as commonly practiced, consists in striking the surface of the area under examination with a view primarily to eliciting sound, and secondarily for the determination of the degree of resistance offered to the percussion blow. During this maneuver the percussion blow may be directed with the finger or with a specially devised percussion hammer, and either directly upon the part under examination, or upon an intervening medium, usually a finger of the opposite hand of the examiner, or in other instances plates of metal, glass, ivory, or hard rubber. The instrument with which the blow is struck, finger or hammer, is termed the *plexor;* the intervening medium, finger or plate, is termed the pleximeter. When an intervening medium or pleximeter is employed, the percussion is termed *mediate percussion;* whereas when no such intervening instrument is present, but the blow is delivered directly upon the part which is under examination, the percussion is termed *immediate percussion.*

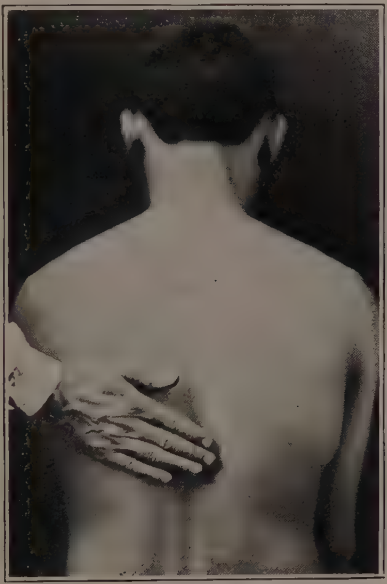


Fig. 165.—Immediate percussion of pulmonary bases.

Immediate Percussion.—In the practice of immediate or direct percussion the pleximeter is dispensed with and the blow is struck directly upon the chest wall. The finger-tips or the palm of the hand may be employed, the former in the percussion of the clavicles to determine the presence of consolidation of the apices of the lungs, and the latter in lightly slapping the two halves of the thorax in the effort to demonstrate the presence of dullness over a relatively large area, usually at the bases posteriorly.

Mediate Percussion.—Mediate, the most commonly employed mode of percussion of the thorax, may be practiced as *instrumental percussion,* or as *finger percussion.* Each method has its advantages and limitations; but it is advisable for the student to perfect himself in one method and to discard the other.

In instrumental percussion the blow is directed upon a pleximeter of rubber, ivory or other material by means of a special percussion hammer. It is essential that the pleximeter shall be sufficiently narrow to fit into the intercostal space without overlapping the adjacent ribs, and it is necessary that the plexor be equipped with a head of soft rubber in order to limit as far as possible adventitious sound occasioned by delivery of the percussion blow. The principal objections to instrumental percussion are that the examiner loses the advantage of variations in the sense of resistance to the percussion blow, which are readily appreciated by the pleximeter in finger percussion, and the adventitious sound which is produced upon striking the hard pleximeter. Likewise, it is difficult to have available pleximeters which fit accurately into the intercostal spaces of all cases under examination.

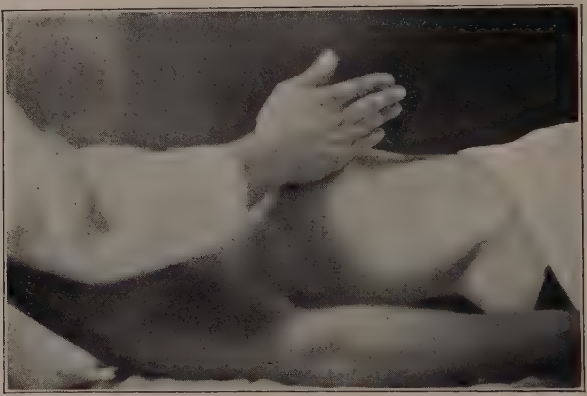


Fig. 166.—Ulnar percussion of upper right quadrant.

In the routine practice of finger percussion the palmar aspect of one finger is applied accurately to the integument in an intercostal space, parallel to the course of the adjacent ribs, while the percussion stroke is delivered by one finger of the opposite hand, the plexor finger being flexed as nearly as possible at a right angle. The stroke is best delivered just back of the base of the nail on the first phalanx of the pleximeter finger which is usually the second finger of the left hand.

The pressure which is exerted by the pleximeter finger depends upon the object of the examination. In practicing superficial percussion with light blows of the plexor, the pleximeter finger should be applied evenly but lightly to the intercostal space; while in the so-called deep percussion with rather forcible blows the pleximeter should be applied more firmly to the integument. In either event, in order to obtain satisfactory results by finger percussion, the blow must be delivered quickly and in a direction perpendicular to the chest wall; the plexor finger must be immediately raised and not be permitted to remain in contact with the pleximeter; the percussion blow must be delivered entirely by a wrist movement, the forearm not participating; and the successive blows must be delivered with uniform intensity.

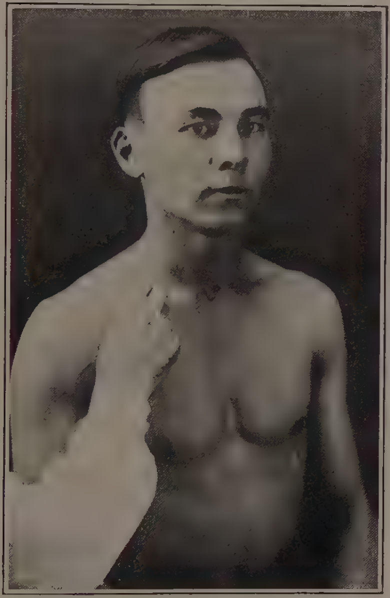


Fig. 167.—Immediate percussion of clavicle.

In practicing finger percussion, only a single blow should be delivered in a given area, as more accurate perception of the sound elicited is obtained in this manner than by a series of blows, which tend to impair the nicety of the auditory appreciation of the tones which are elicited. In comparing the quality of the sound elicited upon the two sides of the thorax, exactly corresponding points should be selected upon either side; and the percussion blows should be delivered with uniform force in each instance.

Certain clinicians prefer to begin their percussion of the thorax above and progress regularly to the inferior regions, while others prefer to start below and work toward the apices of the lungs.

Finger percussion is preferable to instrumental percussion for the reason that the pleximeter finger becomes a sensitive palpating medium, appreciating minor variations in the degree of resistance which the chest wall offers to the precussion stroke. Moreover, the finger may be more accurately applied to the area under investigation, excluding the possible intervention of air between the chest wall and the pleximeter.

Finger percussion is practiced with the most satisfactory results with the patient seated upon a revolving stool or in the standing posture. The room should be comfortably warm in order to prevent fibrillary contraction of the thoracic musculature, and the attitude should be free from muscular tension. The head should be maintained in a symmetrical position in the median line of the body. There is usually a tendency upon the part of the patient to turn the head toward the opposite side during percussion of the superior thoracic regions.

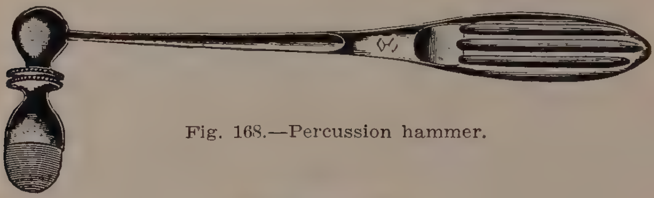


Fig. 168.—Percussion hammer.

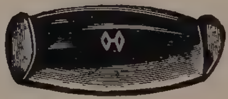


Fig. 169.—Hard rubber pleximeter.

During percussion of the anterior surface of the thorax, the patient should allow the hands to hang naturally at the sides. The examiner should occupy a stool or stand directly in front of the patient, as variations in the quality and pitch of the note elicited cannot be accurately determined if the examiner occupies a position upon one side of the subject. In percussing the supraclavicular and infraclavicular regions in this position, it is desirable that the examiner should by persistent practice become ambidextrous, a degree of proficiency which frequently cannot be attained. In the latter event, the examiner may assume a position upon one side of the patient; and, applying a pleximeter finger evenly to the integument of the supraclavicular and infraclavicular regions, respectively, compare the note elicited upon percussion with blows of uniform intensity above and below the clavicle on the opposite side.

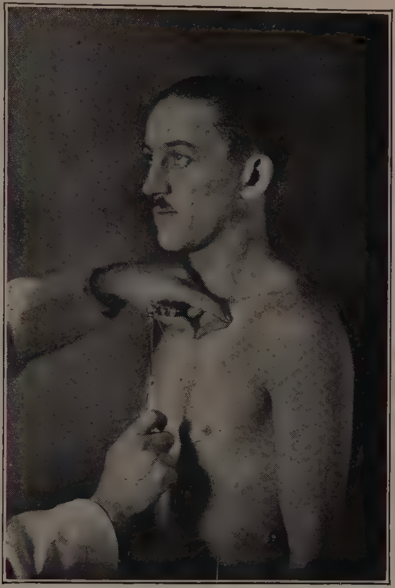


Fig. 170.—Instrumental percussion of thorax.

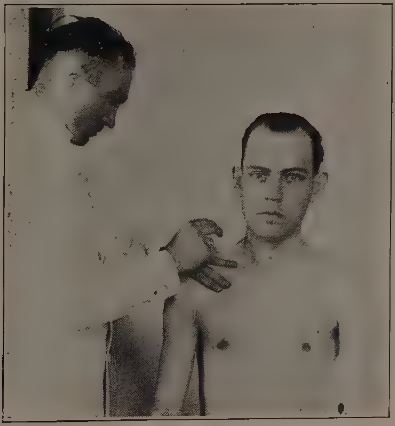


Fig. 171.—Percussion of pulmonary apices.

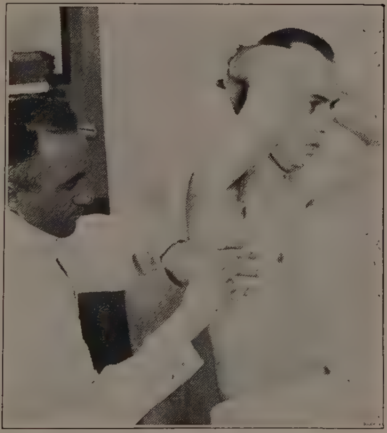


Fig. 172.—Percussion of lateral thoracic region.

During percussion of the lateral regions of the thorax, a good exposure is obtained by directing the patient to raise the arms outward and upward to a horizontal position, or by having him clasp the hands behind the head, the arms meanwhile being drawn backward. The former position is the preferable one, in that it does not put the integument and subjacent muscles in a position of undue tension.

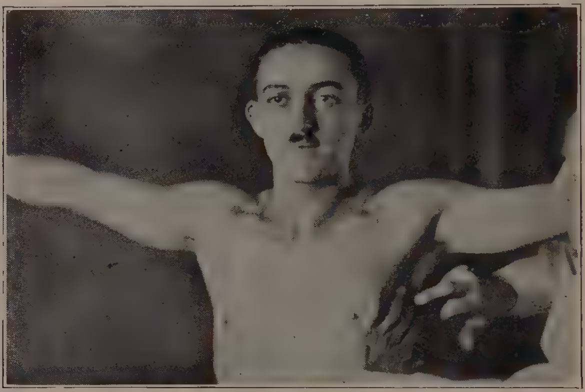


Fig. 173.—Percussion of lateral thoracic wall.

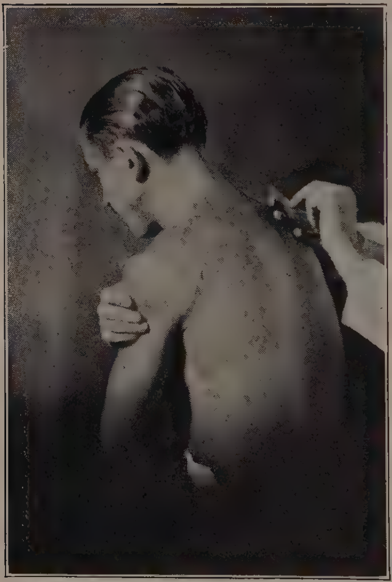


Fig. 174.—Percussion of posterior thorax. Wide separation of scapulæ obtained by clasping hands over opposite shoulders.

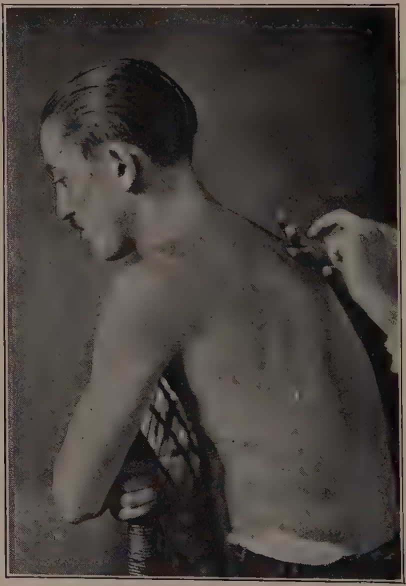


Fig. 175.—Percussion of posterior thorax. Separation of scapulæ obtained by grasping chair-back.

In percussion of the posterior thoracic wall, the patient should bend the trunk slightly forward and clasp each hand over the opposite shoulder, thus insuring a wide separation of the scapulæ and exposure of a considerable area of the posterior thoracic wall. The same end is attained by having the patient straddle a chair and embrace its back with the arms. Percussion of the suprascapular and scapular regions without separation of the scapulæ is devoid of diagnostic value, as the shock of the percussion stroke cannot penetrate the thick beds of muscular and osseous tissue which it encounters under these conditions.

When it is necessary to practice percussion with the patient in the recumbent posture, the examiner should bear in mind the adventitious impairment of resonance in the dependent portion of the thorax when the subject assumes the lateral decubitus. The dulling of the note in this instance may be due to diminution of the aerial content of the dependent lung, or to shifting of the mediastinal structures. When it is necessary to percuss a patient in the recumbent posture, it is essential that he be turned a sufficient number of times to insure the percussion of all regions of the thorax in the most favorable posture obtainable.

PALPATORY PERCUSSION

In the practice of this method of percussion the primary object is the determination of the degree of resistance afforded by the underlying structures rather than the production of sound. Palpatory percussion is employed in both direct and indirect methods. In the former the chest wall is struck with the pads of the finger tips with a pushing movement, the sensitive finger tips noting the degree of resistance to the blow. Indirect or mediate palpatory percussion is practiced by striking the pleximeter finger with a tardy, pushing movement rather than with the sharp, quick stroke of routine percussion, and allowing the plexor to remain for an instant upon the pleximeter.

Palpatory percussion is of service in determining the presence of fluid in the pleural cavity and in outlining the borders of a hypertrophied heart or a large pericardial effusion by the gradations which are encountered in the resistance to the percussion blow.

AUSCULTATORY PERCUSSION

This method of percussion, which combines auscultation with mediate or immediate percussion, is employed for the purpose of outlining the borders of solid organs, consolidations, neoplasms, and collections of fluid. In the practice of auscultatory percussion the chest-piece of the stethoscope is applied over the organ, consolidation, or structure which is to be outlined, and is retained in position by the patient or an assistant while the examiner, after first percussing near the bell of the instrument and fixing in his mind the quality of the note which is elicited, proceeds to percuss toward the instrument from the several directions upon the surface of the thorax. In the performance of this maneuver mediate percussion may be employed or direct palpatory percussion, or immediate percussion in the form of rapid finger flicking. Instead of employing percussion, the same result is obtained if the vibrating tuning-fork is drawn over the integument of the thorax toward the bell of the instrument; or, in the absence of this instrument, by drawing the rubber eraser of an ordinary pencil over the skin.

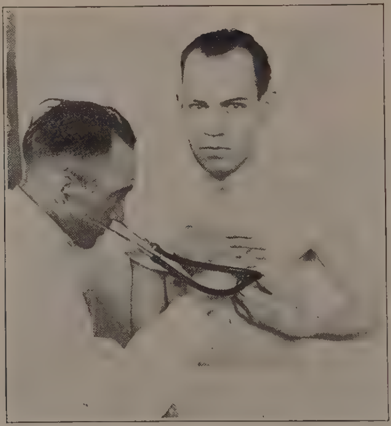


Fig. 176.—Auscultatory percussion.

To obviate the increase in sound volume as the percussion blows approach the stethoscope, arcs of a circle may be drawn on the thorax with a pencil compass or pencil and string. The stethoscope is placed at the central point and the percussion blows struck along the arc, thus the percussion blows are delivered at a given distance from the stethoscope.

In auscultatory percussion of solid structures which are surrounded by pulmonary tissue the note is observed to undergo two successive alterations: a primary impairment of resonance, which is followed secondarily by frank dullness or flatness. The several points at which the percussion note is observed to change may be marked upon the skin with a dermographic pencil; and, when connected by a line drawn through each of them, will indicate the borders of the organ or structure under examination. Most frequently employed in outlining the areas of relative and absolute cardiac dullness, auscultatory percussion is also useful in differentiating by tonal variations between the dullness of the liver and that due to pneumonic consolidation of the right base or pleural effusion of the right side. Similarly, it is an invaluable aid in differentiating consolidations of the left lung from the dullness of the heart.

RESPIRATORY PERCUSSION

This mode of percussion consists in percussion of the two sides of the thorax during quiet respiration and again during forced inspiration with the object of determining minor variations in the pulmonary resonance upon the two sides of the thorax. The method is particularly useful in the detection of minor degrees of consolidation. In incipient phthisis upon consecutive percussion during quiet and during forced inspiration, the dullness upon the consolidated side remains unaltered, while the sound side exhibits an increase of resonance.

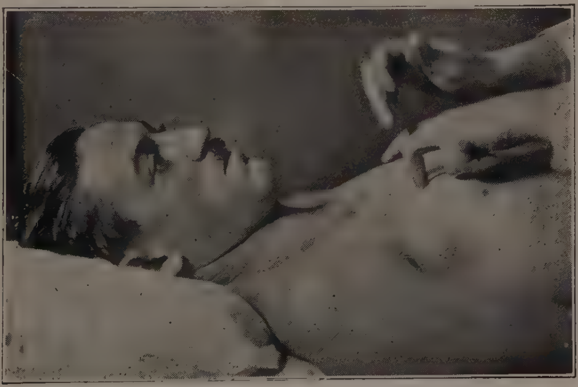


Fig. 177.—Orthopercussion of thorax.

ORTHOPERCUSSION

In the practice of orthopercussion, the pleximeter finger is bent at a right angle at the junction of the first and second phalanges and the tip of the finger is applied to the intercostal space, avoiding contact with the ribs or costal cartilages. The percussion blow is delivered upon the first phalanx, thereby setting up vibrations in a very restricted area of the pulmonary tissues. When the percussion stroke is exceedingly light, the term *threshold percussion* is applied to this method of examination. Threshold percussion is particularly serviceable in delimiting solid organs and effusions of fluid adjacent to air-containing pulmonary tissues.

SUPERFICIAL AND DEEP PERCUSSION

The terms superficial and deep as applied to percussion have special reference to the force of the percussion blow, and each method has its indications in the examination of the thoracic viscera. In practicing superficial percussion the thorax is tapped quickly and lightly, whereas in deep percussion the blow is delivered with sufficient force to set up vibrations in the deeper structures. In the examination of persons with thin chest walls as well as in the examination of children, superficial percussion is to be employed. It is also to be preferred in the delimitation of consolidations which are situated immediately beneath the chest wall, in order to obviate the establishment of confusing vibrations in the deeper structures.

In the examination of patients with thick chest walls, on the contrary, and in the elicitation of dullness of deep consolidations which are covered by normal pulmonary tissue, forcible or deep percussion is to be employed.

Deep or forcible percussion has a very restricted field of usefulness in physical exploration of the thorax. A forceful blow against the chest wall immediately creates so much lateral vibration that the sound produced by the structures immediately subjacent to the pleximeter is obscured to some degree. Moreover, it has been estimated that with relatively forcible percussion blows the sphere of the vibrations does not exceed 6 centimeters in depth; hence, it is obviously impossible by deep percussion to reveal the presence of a central pneumonia or a solid lesion which is situated deeply within the thorax.

DEGREE OF RESISTANCE

Aside from eliciting sound, percussion is employed to determine the degree of resistance as appreciated by the pleximeter. In many instances the degree of resistance encountered affords as valuable information as does the sound which is elicited; and in cases where for any reason the sounds produced are not typical, it may be the sole guide of the examiner. Increased resistance to the percussion blow as appreciated by the pleximeter indicates a decrease in the air content of the part and a corresponding increase in solid structure or the presence of fluid. A high-pitched note with well-marked sense of resistance indicates that the air content is small while the proportion of solid material is correspondingly in excess of the normal amount.

Piorry, Traube, and Ebstein have each in turn emphasized the importance of the degree of resistance in chest diagnosis, and in recent years Pottenger has carried this phase of physical diagnosis to a high point of perfection.

ATTRIBUTES OF THE PERCUSSION SOUND

The sound which is elicited upon mediate percussion of the thorax is derived from three sources; namely, the pleximeter, the thoracic wall, and the tissues which are subjacent to this wall and within the sphere of the shock of the percussion blow. Hence, the character of the note varies with the force of the stroke which is employed and with the region of the thorax which is under examination. In all cases the examiner should study the quality, pitch, intensity, and duration of the note which is elicited.

Quality.—Quality is the property or attribute by which a given sound is distinguished from a sound of different origin. It is by the quality of the sounds that the sound which is produced upon striking a piece of iron is distinguished from that which is produced upon striking a piece of wood, and by which the sound which is produced upon percussing over the normal lung is differentiated from that which is elicited upon percussing over a solid organ. It is their quality which gives to the various sounds which are elicited upon percussion their individuality and much of their localizing value.

Pitch.—The pitch of a sound is determined by the rapidity of the vibrations by which the sound is produced. When the surface of the thorax overlying the lung is percussed, the air content of the innumerable pulmonary alveoli is thrown into vibration. Pitch may be high or low, depending as it does upon the rapidity of these vibrations, rapid vibrations producing a note of high pitch, while slow vibrations produce one of low pitch. The rate of the vibrations is in turn influenced by the size of the cavity containing the air and by the force of the percussing blow, being more rapid in small cavities and with forcible percussion blows.

Intensity.—Intensity, or volume, has reference to the loudness of the sound, this in turn depending upon the amplitude of the vibrations which are generated, the force of the percussion blow, the thickness of the chest wall, and the amount of air in the area under examination. With heavy percussion over an area of lung containing an excess of air and with a thin chest wall, an intense sound is produced; while a similar stroke over a region containing little air, overlaid by thick chest walls, produces a sound of minor intensity.

Duration.—The duration of the percussion sound possesses less diagnostic significance than do the other attributes. In general, a dull note is of shorter duration and higher pitch than is a frankly resonant note.

CHAPTER XXIII

PERCUSSION OF THE LUNGS

NORMAL PERCUSSION SOUNDS

Pulmonary Resonance.—The sound which is elicited upon percussion of the surface of the thorax which overlies normal crepitant pulmonary tissue is termed *pulmonary resonance,* or *normal vesicular resonance.* When the thoracic surface is percussed, the aerial content of the pulmonary infundibula and alveoli which are situated within the range of the shock of the percussion blow is caused to vibrate, with the consequent production of sound. The quality of this sound is distinctive, and is only afforded by percussion of normal pulmonary tissue containing its normal quota of air separated by innumerable interalveolar septa. The intensity and pitch of the sound depend upon the amplitude and rate of the vibrations which are generated in the aerial content of the closed spaces, by the distance of these air-containing spaces from the thoracic surface, and by the thickness of the chest walls. With strong percussion, vibrations of greater amplitude are generated with the production of a more intense sound than that which is produced by feeble percussion. Similarly percussion of the thorax in the infraclavicular and axillary regions, situations in which the pulmonary tissue is closely apposed to the thoracic walls is attended by a more intense sound than is percussion of the region of the shoulder girdle where the lung is separated by voluminous musculature and bony structures from the surface of the thorax. Similarly in the thin thorax of the female subject and the child the resonant note of percussion of the normal lung possesses greater intensity than in the corresponding region in the normal adult male subject.

The normal thorax presents certain *regional variations* in pulmonary resonance with which it is essential that the examiner be familiar. Pulmonary resonance of maximum purity is obtained upon percussion of the first two intercostal spaces in the infraclavicular region for the reason, as Seitz has shown, of the greater width of the intercostal spaces, and the relative thinness of the thoracic wall in this region. In these interspaces, however, the resonance exhibits minor variations in its intensity. It attains its maximum intensity in the midclavicular line, from which point it diminishes in intensity as one percusses outward toward the shoulder where the great pectoral muscle becomes more voluminous, and inward toward the sternum, in which situation the anterior border of the lung becomes progressively thinner, and presents a less voluminous bed of pulmonary parenchyma in the sphere of the shock of the percussion blow. In right-handed subjects the resonance is usually slightly less intense upon the right side of the thorax in the first two interspaces, owing to the more excessive development of the pectoral muscles upon this side. It is, moreover, frequently noted that the resonance which is elicited in the first intercostal space is slightly less intense than that which is produced upon percussion of the second interspace, a fact which is explained by the fact that the first intercostal space is more deeply situated than is the second owing to the backward course of the first rib beneath the clavicle, and by the fact that in the act of mediate percussion the pleximeter is forced in the majority of cases to rest partially upon these bones.

During percussion of the first two intercostal spaces, careful comparison of the intensity of the resonance should be made upon the two sides of the thorax. In percussion of the regions of the anterior thoracic surface below the second interspace such comparison is useless on account of the influence which the projection of the heart toward the left has upon the intensity of the two sounds.

In the third and fourth intercostal spaces the pulmonary resonance is less pure than in the first two spaces, owing to the extensive distribution of the great pectoral muscle in these regions, to the thickness of the panniculus adiposus of the mammary gland, and, upon the left side, the projection of the heart to the left of the sternal border. Seitz holds that the more pronounced narrowing of the intercostal spaces as one descends upon the anterior thoracic wall plays an important part in the progressive enfeeblement of pulmonary resonance which is encountered

In the fifth intercostal spaces pulmonary resonance is variable upon the two sides of the thorax. Upon the left side of the thorax in the male subject the intensity of the resonance is moderately impaired by the presence of the mammary gland, and markedly impaired by the same gland in the female subject. Moreover, internal to the midclavicular line upon this side of the thorax in the area of relative cardiac dullness, corresponding to the region in which the heart is invested by the anterior pulmonary border, which becomes progressively less voluminous, there is marked impairment of pulmonary resonance. Similarly, upon the right side of the thorax in the fifth interspace the area of relative hepatic dullness is encountered in which the upper diaphragmatic surface is clothed by the thin lower border of the lung, yielding a diminution of resonance which is soon to terminate in frank flatness when the area is attained in which the liver is directly apposed to the anterior thoracic wall. In the right fifth intercostal space the areas of relative cardiac and relative hepatic dullness meet at almost a right angle, forming an angle of pulmonary resonance immediately to the right of the sternum in this interspace, *Ebstein's cardiohepatic angle.*

In the sixth right intercostal space pulmonary resonance exhibits an abrupt transition into flatness, indicating in this situation the superior limit of the area of hepatic flatness, in which the liver is in immediate apposition, beneath the diaphragm with the anterior thoracic wall.

In all regions of the thorax percussion over the ribs and bony structures yields pulmonary resonance of less purity than that which is obtained upon percussion of the intercostal spaces. Indeed, as one percusses inward in the superior intercostal spaces toward the sternum, as this bone is encountered, the *osteal resonance* afforded by percussion of this bone blends with the pulmonary resonance of the intercostal spaces.

In the supraclavicular regions pulmonary resonance is encountered which becomes progressively more intense as one percusses inward toward the median line, to finally give place in this location to the tympany of the trachea. The progressive increase in intensity of pulmonary resonance under these circumstances should be in all cases attributed to its real cause, the proximity of the trachea to the pulmonary apices in the median line of the body. Of quite different significance is hyperresonance encountered in these regions, when this is encountered throughout the region and extends well outward toward the distal extremity of the clavicle.

Percussion of the clavicle yields resonance of impaired intensity, which contrasts markedly with the purity of the pulmonary resonance in the infraclavicular region. Excessive curving of this bone further impairs the purity of the resonance as do also irregularities due to former fractures or osseous disease.

Percussion of the sternal region, overlying the sternum and ensiform cartilage, yields three distinct variations in pulmonary resonance. Aside from the influence which the osteal resonance of the bone exerts upon the pulmonary resonance, there are distinct variations in the quality and intensity of the pulmonary resonance as a result of the influence upon the latter of the anatomic structures with which the sternum is in relation in different portions of its distribution. Percussion of the manubrium sterni and the portion of the gladiolus above the level of the fourth costal cartilage yields upon forcible percussion resonance blended with the tympany of the subjacent trachea and primary bronchi. Below this level percussion of the gladiolus yields rather marked impairment of resonance, corresponding to the area of relative cardiac dullness in which the heart is covered by the thin borders of the lungs. Percussion of the ensiform cartilage yields frank dullness or flatness, on account of its apposition with the left lobe of the liver. However, in the presence of extreme distention of the stomach with gas, this flatness may be replaced with gastric tympany, which has nothing in common with the quality of pulmonary resonance.

Upon the lateral walls of the thorax pulmonary resonance is somewhat less intense than upon the anterior wall; but it is decidedly more intense than is the case upon the posterior wall of the thorax. In this regard, the three aspects of the thorax present the same relative variations in regard to the intensity of pulmonary resonance as was observed in the same regions in the study of vocal fremitus.

In the case of vocal fremitus the tactile vibrations are observed with slightly exaggerated intensity upon the right lateral wall of the thorax, in the case of pulmonary resonance the reverse obtains; pulmonary resonance is slightly diminished upon the right lateral thorax, a diminution which is caused by the same factors which make for increased vocal fremitus in this area.

In the axillary region pulmonary resonance is slightly less intense than in the infraaxillary region; but in the upper axillary region the note is sometimes slightly tympanitic on account of the closer proximity to the chest wall in this region of the larger bronchi. Upon percussion of the lateral thoracic wall the resonance is observed to augment in intensity as one descends until the seventh intercostal space is reached. At this level upon the right side of the thorax pulmonary resonance is impaired by the advent of the area of relative hepatic dullness; while at the same level upon the left side of the thorax, it is replaced by the tympanitic sound generated in the stomach and colon.

In every portion of the posterior surface of the thorax pulmonary resonance is less intense than upon the anterior and lateral regions of the thorax. Upon the posterior thoracic wall resonance of greatest purity is obtained upon percussion of the pulmonary bases in the infrascapular region. Next in order of purity comes the resonance in the inferior half of the interscapular region, and this is followed by the superior half of the same region. In the suprascapular and scapular regions, overlying the bony scapulæ with their thick investment of musculature, pulmonary resonance is less intense than in any other portion of the posterior thoracic surface. In this region the minimum intensity is encountered along the course of the spine of the scapula.

The Normal Limits of Pulmonary Resonance.—Anteriorly and inferiorly the normal limits of pulmonary resonance correspond accurately to the anatomic site of the borders of the lungs. On the ventral surface of the thorax the limits of resonance lie behind the sternum in the infraclavicular and upper mammary regions, the osteal resonance of this bone interfering greatly in the delimitation of the anterior pulmonary borders in this region. At the level of the fourth intercostal space, however, the anterior limit of resonance is displaced to the left by the interposition of the heart between the left lung and the chest wall, in the area of absolute cardiac dullness.

The inferior limits of normal pulmonary resonance are encountered at the level of the sixth rib in the midclavicular line, the eighth rib in the midaxillary line, and the tenth rib in the scapular line.

Krönig in 1907 demonstrated that the normal area of pulmonary resonance in the supraclavicular and suprascapular regions, instead of conforming to the anatomic apex of the lung, which on either side occupies a position between the sternal and clavicular heads of the sternomastoid muscle, is represented by a zone or isthmus possessing well-defined limits in the normal subject. The narrow central portion of this zone, corresponding to the level of the shoulder girdle, measures ordinarily 5 centimeters or 2 inches in width.

Krönig's isthmus is bounded internally by a line drawn from the sternoclavicular articulation upward with its concavity directed outward in the lower supraclavicular region, inward in the cervical region, and again outward in the suprascapular region, where it terminates at the level of the second thoracic spine. The external limit of this zone of resonance is bounded

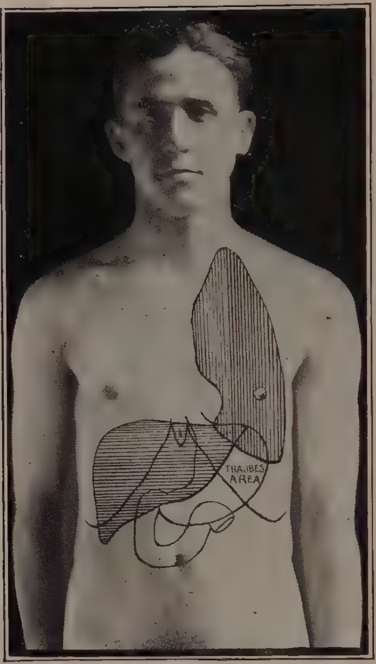

TRAUBES AREA

Fig. 178-*A*.—Traube's semilunar space.

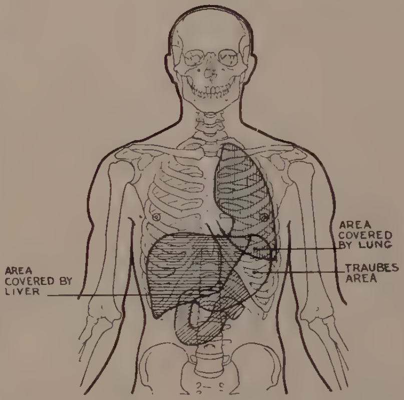

AREA COVERED BY LUNG
TRAUBES AREA
AREA COVERED BY LIVER

Fig. 178-*B*.—Traube's semilunar space.

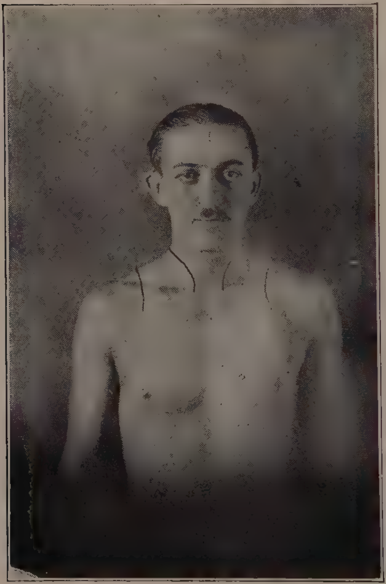


Fig. 179.—Krönig's resonant zones (anterior view).

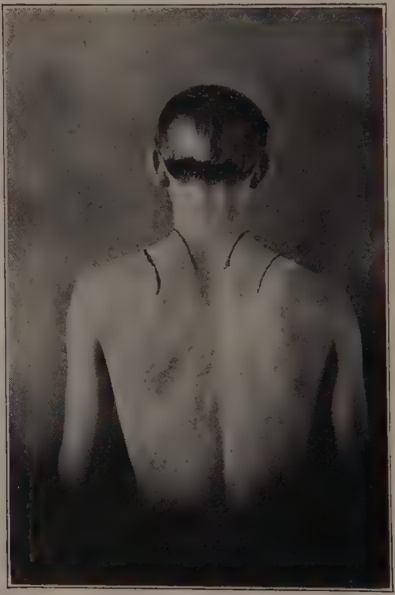


Fig. 180.—Krönig's resonant zones (posterior view).

by a line drawn from the junction of the outer and middle thirds of the clavicle anteriorly, with its concavity directed outward, terminating at the midpoint of the scapular spine posteriorly, with its concavity likewise directed outward toward the shoulder.

Variations in the Limits of Pulmonary Resonance.—In bronchopulmonary disease any lesion which increases the expansion of the lung in any direction results clinically in a corresponding increase in the area of pulmonary resonance; and any lesion which decreases the extent of the lung in any direction produces a corresponding limitation of the resonance to the extent of the lesion.

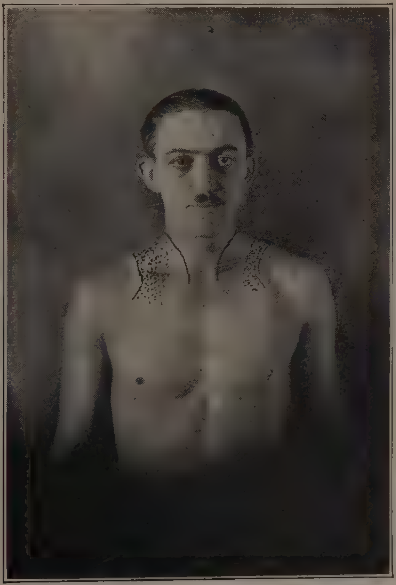


Fig. 181.—Impairment of resonance in Krönig's zones.

It follows that in disease of the lung or pleura there may be a general increase or decrease of resonance at all of the pulmonary borders; that the variation may involve two corresponding borders; or that only a single area of the lung may present an abnormal variation in the extent of pulmonary resonance.

A general increase of pulmonary resonance in all directions, all of the borders of the lungs occupying a position beyond their normal limits, is characteristic of hypertrophic emphysema and is noted during the paroxysm of bronchial asthma. A similar general increase is frequently noted in dyspneic patients with uncompen-

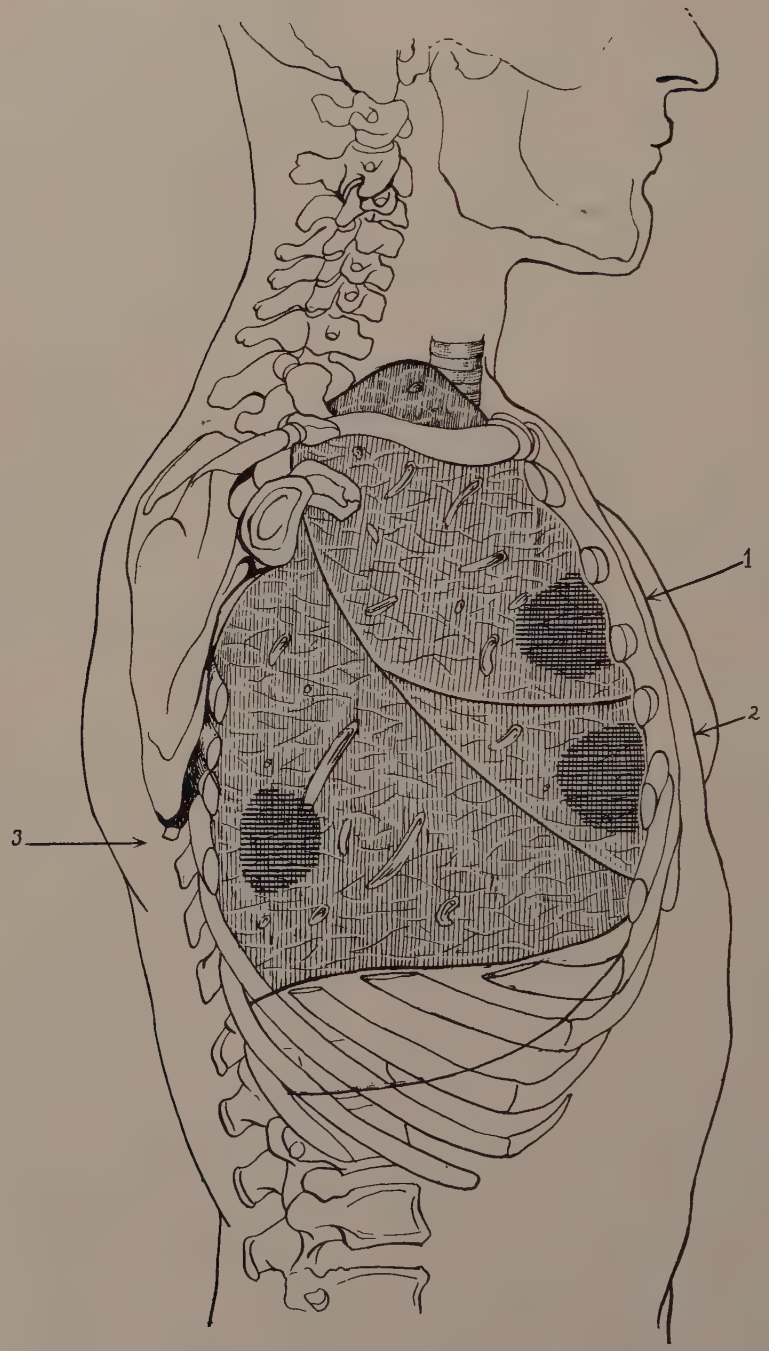


Fig. 182.—Physical causes of change in percussion note. *1-2*, dullness upon percussion; *3*, deep dullness masked by intervening lung.

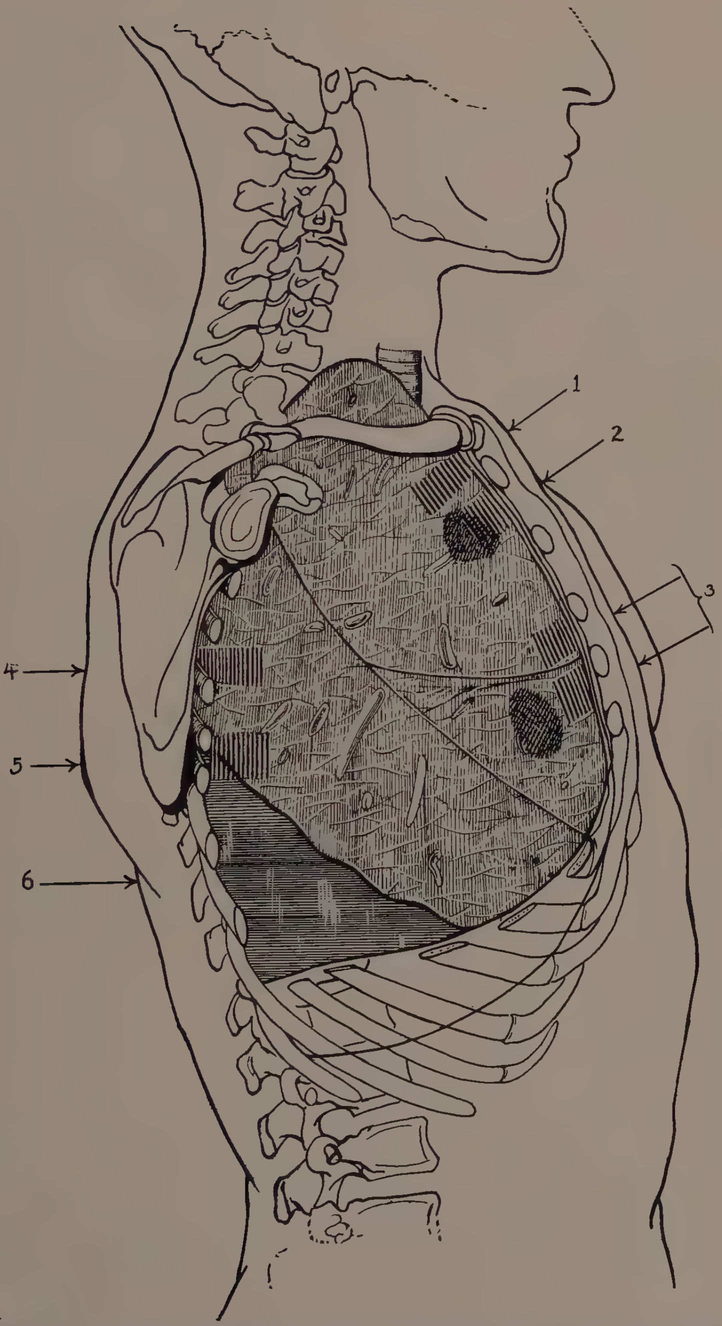


Fig. 183.—Physical causes of change in percussion note. *1*, normal vesicular resonance; *2-3*, impaired resonance, or relative dullness; normal vesicular resonance upon superficial percussion, impaired resonance upon deep percussion; *4*, normal vesicular resonance; *5*, dullness; *6*, flatness of fluid origin.

sated heart disease, and occasionally in subjects of chronic bronchitis.

A general decrease of pulmonary resonance, due to general retraction of the pulmonary borders, occurs with atrophic emphysema, in which the lungs are symmetrically shrunken and atrophic.

Extension of resonance at the apices occurs in connection with the paroxysms of bronchial asthma and pertussis as a result of the acute vesicular emphysema which attends these attacks. When hypertrophic emphysema involves the apices extensively, there is extension of the limits of resonance in the supraclavicular regions.

Decreased resonance at the apices, as evidenced by a narrowing of Krönig's isthmus, occurs with chronic ulcerative and fibroid phthisis, apical pneumonia, pulmonary collapse from bronchial obstruction, and as a result of traction of adhesions in chronic adhesive pleurisy. The apical limitation of resonance in phthisis is frequently bilateral, whereas that of apical pneumonia and chronic adhesive pleurisy commonly involves one apex.

Extension of resonance of the anterior borders of the lungs so that they encroach upon or obscure the area of cardiac dullness occurs in established hypertrophic emphysema. During the transient acute vesicular emphysema of the paroxysm of bronchial asthma and of pertussis the anterior border of the left lung encroaches upon the area of cardiac dullness.

Decreased resonance of the anterior border of one or both lungs is indicative of fibroid retraction of the lung in chronic interstitial pneumonia or fibroid phthisis or of displacement of the pulmonary border by a hypertrophied heart or by pericardial or pleural effusion. In the case of fibroid retraction of the left lung the cardiac impulse is diffuse, occupying a wide area in the third, fourth, and fifth interspaces; whereas in the presence of pleural or pericardial effusion the impulse is displaced or is invisible.

Increased resonance of the lower border of the lungs is part and parcel of the general extension of resonance accompanying hypertrophic emphysema or bronchial asthma. It may also occur with fibrinous bronchitis and uncompensated heart disease.

Decreased resonance of the lower borders of one or both lungs points to fibroid retraction of the lung due to chronic interstitial pneumonia or fibroid phthisis; or to elevation of the diaphragm due to paralysis of that muscle or to the subphrenic pressure of ascites, abdominal tumor, hepatic, or splenic enlargement. In atelectasis and in pleurisy with effusion the lower border of the lung is likewise elevated with diminution in the area of pulmonary resonance in this direction.

The Respiratory Excursion of the Lung.—In estimating the respiratory excursion of the lungs, the lower limit of pulmonary resonance should be defined by percussion in the midclavicular, midaxillary, and scapular lines during quiet and during forced inspiration, and the difference noted. Usually measuring approximately one inch, the respiratory excursion is very much reduced in subjects of hypertrophic emphysema and during asthmatic attacks, as well as in cases of limited diaphragmatic excursion occurring as a result of increased subphrenic pressure or painful thoracic affections.

ABNORMAL PERCUSSION SOUNDS

Whereas the sound elicited upon percussion of the thorax over normal pulmonary tissue possesses a distinctive resonance of a relatively low pitch and considerable duration, when the aerial content of the lung is diminished by solid or fluid elements the note elicited is of higher pitch, shorter, and rather unmusical. The diminution or absence of the resonant quality has led to the designations *impaired resonance, relative dullness, dullness,* and *absolute dullness* or *flatness,* according to the degree of impairment of the sound.

Similarly, when the pulmonary parenchyma contains an excess of air or is the seat of excavation, the sound which is elicited upon percussion possesses an added resonance, constituting *hyperresonance* or *tympany,* depending upon the degree of resonance of the note elicited.

Impaired Resonance.—The percussion note exhibits slight impairment of the purity of vesicular resonance, not amounting to dullness, which is the next gradation, when there is only a moderate increase in the solid over the normal crepitant structure of the lung. Impaired resonance is elicited particularly in the early stages of phthisis at the apices of the lungs. Impairment of resonance is also a sign of moderate pleural thickening and incipient consolidation from pneumonia or other cause. It is the first step toward dullness, but is not so pronounced in its change of quality.

The note which is elicited upon percussion over airless structures which are separated from the chest wall by the intervention of normally crepitant lung is termed *relative dullness.* This note is elicited in percussion of the areas of relative cardiac and relative hepatic dullness, in which these organs are overlapped by the borders of the lungs.

Dullness.—As in the case of impaired resonance, dullness indicates a decrease in the air-content of the part and a corresponding increase in the solid elements in the area under examination; but the note is more materially altered than it is in impairment of resonance.

Flatness.—Flatness, a note which is entirely devoid of resonance, is indicative of the absence of air from the area within the sphere of the percussion blow. Frequently the transition from a marked degree of dullness to flatness is detected with considerable difficulty.

The degree of dullness encountered in any region of the thorax is dependent upon the volume of air in the parts percussed, their distance from the thoracic wall, and the force of the percussion blow. In general, the more dense a consolidation, and the more nearly it approaches the thoracic wall, the greater the degree of dullness which is elicited. On the other hand, a patch of consolidation which is separated from the chest wall by a band of crepitant pulmonary tissue may fail to produce a dull note upon light percussion, as the shock of the blow may fail to reach the area of airless tissue. However, attempts to demonstrate such a patch of consolidation by forcible percussion frequently fail on account of the numerous vibrations which are set up in the surrounding aerated pulmonary alveoli, whose resonance effectually masks the dulling of the note which might be produced by the area of consolidation. Similarly, in dealing with a small area of consolidation which is closely subjacent to the thoracic wall, but which is surrounded by air-containing pulmonary tissue, the percussion must be exceedingly light in order to avoid setting up vibrations in the neighboring alveoli, which would effectually obscure the dullness occasioned by the small consolidated area. Very frequently in dealing with the disseminated patches of consolidation in bronchopneumonia it is impossible even by light percussion to demonstrate dullness on account of the compensatory hyperresonance of the vicariously inflated adjacent alveoli of the lung.

Over areas of the thorax where dullness or flatness is elicited, careful finger percussion will usually show that the sense of resistance to the pleximeter finger is distinctly increased.

Dullness is elicited upon percussion of the thorax in the presence of the consolidations of lobar pneumonia and phthisis, infiltration of the lung with edema and hypostatic congestion, in carcinomatous infiltration, in the presence of considerable degrees of pleural thickening, in the area occupied by a superficial pulmonary neoplasm, in atelectasis which is extensive in distribution, and in pulmonary syphilis and hemorrhagic infarction of the lung when favorably situated, pathologic conditions in which the aerial content of the lung is partially replaced by solid or liquid elements.

The distribution of the area of dullness or flatness possesses distinct diagnostic significance in many cases. Apical dullness points in the majority of cases to chronic phthisis, particularly if it involves the right apex. An equalization of the resonance over the two upper lobes is likewise suggestive of phthisis, as the upper right lobe normally exhibits a slight degree of impairment of resonance.

Parasternal dullness occupying the lower infraclavicular or upper mammary region may be due to mediastinal tumor of extensive dimensions. In this event localizing pressure signs are commonly in evidence.

Impairment of the resonance in Traube's semilunar space, commonly attaining to the degree of flatness, may be due to a left-sided pleural effusion, or it may be caused by subphrenic abscess, or by splenic or hepatic enlargements. Similarly, dulling of the pulmonary resonance in Ebstein's cardiohepatic angle attends right-sided pleural effusion, or an extensive pericardial effusion, constituting *Rotch's sign* of the latter disease.

Circumscribed dullness to the left interscapular region is occasionally to be noted in connection with aneurysm of the thoracic aorta. Dullness in this region may, however, signify enlargement of the bronchial glands or mediastinal tumor.

Serofibrinous pleurisy with abundant effusion and empyema are attended by flatness over an extensive area of the lower posterior or lateral region of the thorax, with frequently a triangular area of paravertebral dullness upon the side opposite to the effusion, occupying the level of the twelfth dorsal vertebra, constituting *Grocco's sign* of these diseases. This dull area is attributed to shifting of the mediastinal structures, occasioned by pressure of the fluid in the opposite pleural cavity.

Hyperresonance.—Hyperresonance is lower pitched than normal vesicular resonance, and occupies a point acoustically be-

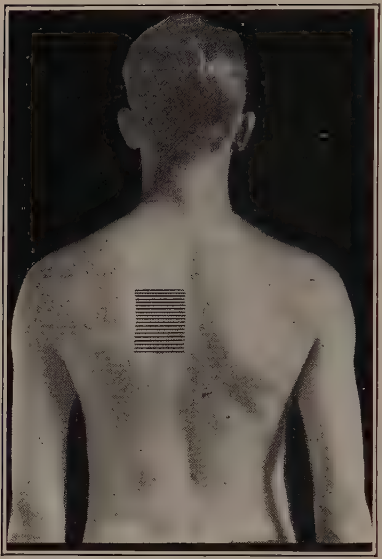


Fig. 184-*A*.—Dullness of aortic aneurysm.

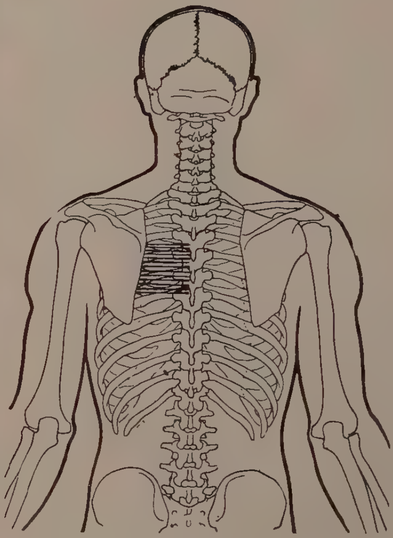


Fig. 184-*B*.—Dullness of aortic aneurysm.

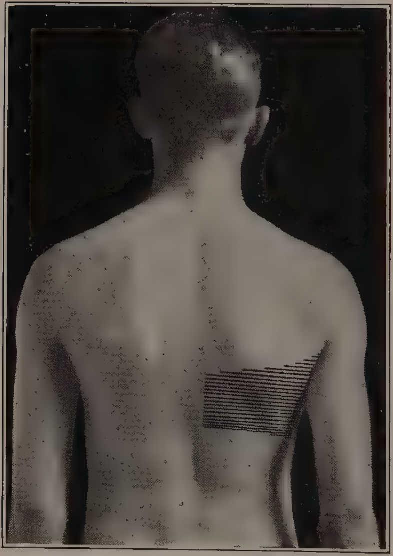


Fig. 185-*A*.—Area of dullness in moderate pleural effusion.

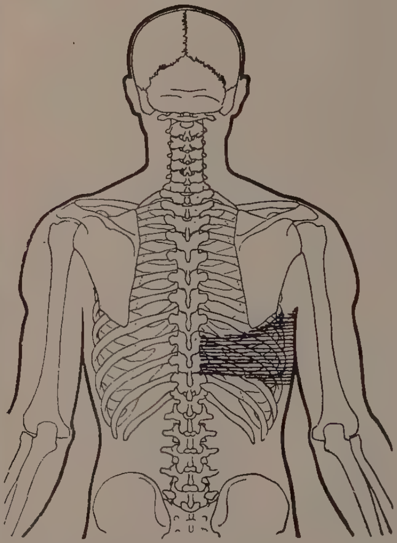


Fig. 185-*B*.—Area of dullness in moderate pleural effusion.

tween normal resonance and tympany. It is elicited upon percussion of the thorax when the subjacent pulmonary alveoli are in a state of partial relaxation from partial compression (Skodaic resonance), or overdistention, as in emphysema or bronchial asthma. In aged subjects, with the atrophy and relaxation of the pulmonary tissues, a hyperresonant note is frequently elicited without other evidence of emphysema.

Hyperresonance during health is encountered in the young child, where the very elastic lungs are readily subject to extreme inflation from respiratory overaction, and where the thoracic wall does not attain great thickness. The marked exaggeration of resonance encountered under these circumstances has been designated "bandbox resonance."

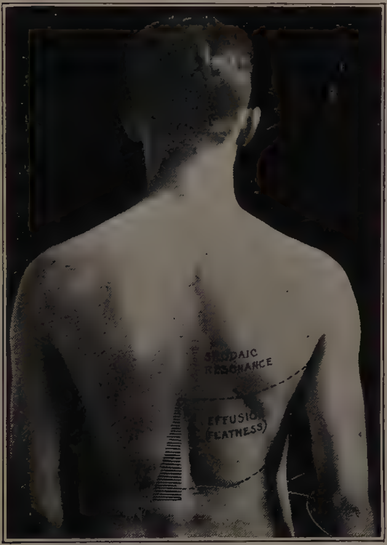


Fig. 186-*A*.—Percussion findings in serofibrinous pleurisy with effusion.

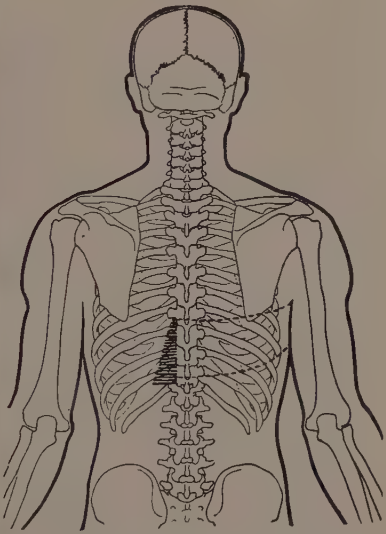


Fig. 186-B.—Percussion findings in serofibrinous pleurisy with effusion.

Pathologically, pulmonary hyperresonance may be bilateral, unilateral, or localized to a limited area of the chest. When bilateral hyperresonance is elicited, it usually indicates hypertrophic emphysema; when unilateral, compensatory emphysema is the usual underlying cause; while localized hyperresonance is commonly elicited over a limited portion of a lung which is the seat of vicarious expansion to compensate for an adjacent focus of cavitation, collapse, compression or consolidation.

Hyperresonance elicited over the median portion of the supraclavicular region is usually to be attributed to the proximity of the air-containing trachea; but it should be borne in mind that a deeply situated pulmonary cavity may yield a note upon percussion simulating hyperresonance.

TYMPANY

Tympany represents the acme of hyperresonance, percussion of the thorax yielding in this instance a tympanitic or drum-like sound. Its prototype in the human economy is produced by percussion over the stomach when this viscus is moderately distended with gas and by percussion over the larynx and trachea. Tympany is encountered upon percussion over pulmonary cavities which contain air, whether these possess a patent bronchial communication or are closed.

The intensity of the tympanitic note depends upon the amplitude of the vibrations which are generated within the area percussed, upon the distance of the area in question from the thoracic walls, and upon the thickness of these walls. The intensity of the sound is greater in the case of large superficial cavities than in the case of cavities of smaller size owing to the greater mass of air which is set in vibration and the readiness with which the vibrations are transmitted to the exterior of the thorax.

The pitch of the tympany depends upon the size of the cavity; upon the presence or absence of a patent bronchial communication; upon the diameter of the bronchial communication when such is present; and upon the tension of the walls of the cavity. In interpreting variations in the pitch of the tympanitic sound in any case the student should bear in mind the physical principles governing the pitch of the tympanitic sound as generated by percussion over pulmonary excavations; namely, that the pitch of the tympany is in inverse proportion to the length of the column of air which is set in vibration upon percussion of the cavity, and in direct proportion to the diameter of the bronchial communication, if there be one; that the shorter the air column which is set in vibration, or in other words, the smaller the dimensions of the cavity, the higher is the pitch of the tympany; that the greater the diameter of the bronchial communication, the higher is the pitch, and the smaller the transverse diameter of the bronchial communication, the lower is the pitch; that of two *closed* cavities the tympany will be of higher pitch in the one which shall have the lesser vertical diameter, and vice versa; and that percussion of a large closed cavity may yield a tympanitic note of higher pitch than a smaller cavity which has a bronchial communication of small diameter, the lowering of the pitch in the latter instance being determined by the narrow orifice of the bronchial communication. From this last consideration it follows that before basing conclusions as to the probable dimensions of a pulmonary cavity upon the pitch of the tympany, it is necessary to establish whether the cavity is closed or open by the changes of sound of Wintrich and Friedreich, to be described in a subsequent paragraph.

The mechanism of generation of the tympanitic percussion sound and its quality when generated are in very intimate relationship the one with the other. Upon percussion of a pulmonary cavity with smooth walls vibrations are created in the air of varying amplitude. In the case of cavities with smooth walls and with free bronchial communication the state of affairs is in some degree analogous to that which obtains in the pipes of an organ or other wind instrument in which the quality and pitch of the notes depend upon variations in the length of the vibrations which are produced. Like the pipe of the organ, a pulmonary cavity with smooth walls and free bronchial communication acts as a resonator for certain of the vibrations which are created in the air content upon percussion. The quality of the tympany which is elicited depends upon the rate of vibration of the fundamental vibrations, which are the slowest vibrations which the chamber is capable of responding to, and the rapidity of these vibrations is in direct relation to the length of the column of air contained in the cavity, or, in other words, to the length of the cavity. It is thus that is explained the change of sound of Wintrich, which will be discussed in a subsequent paragraph.

It is essential that a cavity possess reasonably smooth walls in order to yield a tympanitic sound upon percussion; and when the walls of a cavity become coated with fibrinous exudate or tissue débris, the tympanitic character of the sound elicited upon percussion is diminished or lost. Wintrich found in his experiments that upon percussion above the orifice of a cylinder sunk in soft, dry snow that he obtained tympany, whereas upon percussion above the ragged depression left after removal of the cylinder that tympany was absent. He also demonstrated the absence of tympany upon percussing above vessels of leather, felt, and other materials of irregular surface.

A pulmonary cavity must attain certain dimensions in order to yield a tympanitic percussion sound. Wintrich places the minimum diameter at six centimeters; while Skoda failed to obtain tympany in cavities approximately the size of the fist.

While all pulmonary cavities which contain air and possess a patent bronchial communication present the physical requirements for the production of tympany upon percussion, it frequently happens that cavities are too deeply seated in the lung to yield tympany upon percussion. It is only when such a cavity is situated in the periphery of the lung that tympany is to be elicited, and even in these cases cavitation is frequently not detected by the experienced examiner.

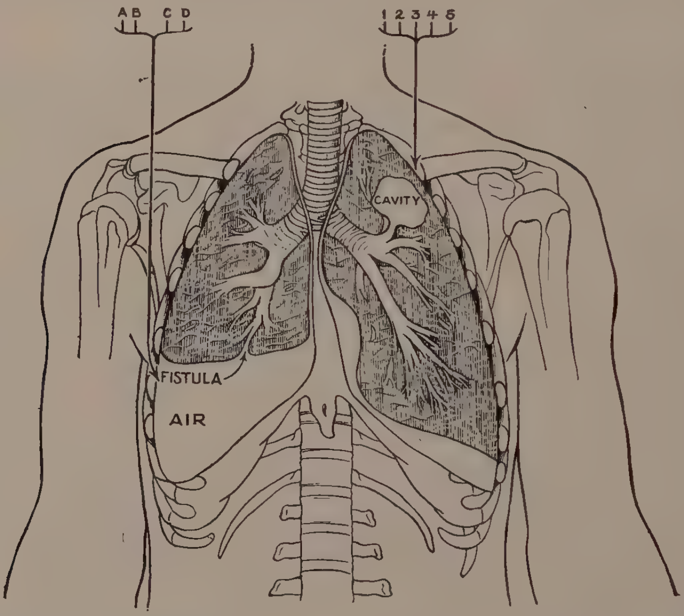


Fig. 187.—Physical basis of pathologic physical signs upon percussion and auscultation of the thorax.

A, cracked-pot sound; *B,* Biermer's phenomenon; *C,* pectoriloquy; *D,* lung-fistula sound; *1,* cracked-pot sound; *2,* Wintrich's change of sound; *3,* Friedreich's change of sound; *4,* bronchial or amphoric respiration; *5,* whispering pectoriloquy.

The tympanitic percussion sound is capable of exhibiting four changes of sound under appropriate conditions; namely, the *change of sound of Wintrich,* the *interrupted change of sound of Wintrich,* the *respiratory change of sound of Friedreich,* and the *change of sound of Gerhardt.*

WINTRICH'S CHANGE OF SOUND

Wintrich demonstrated that upon percussion of a superficial pulmonary cavity with patent bronchial communication the tympany is of higher pitch with the patient's mouth open and of lower pitch when the lips are tightly closed during the percussion.

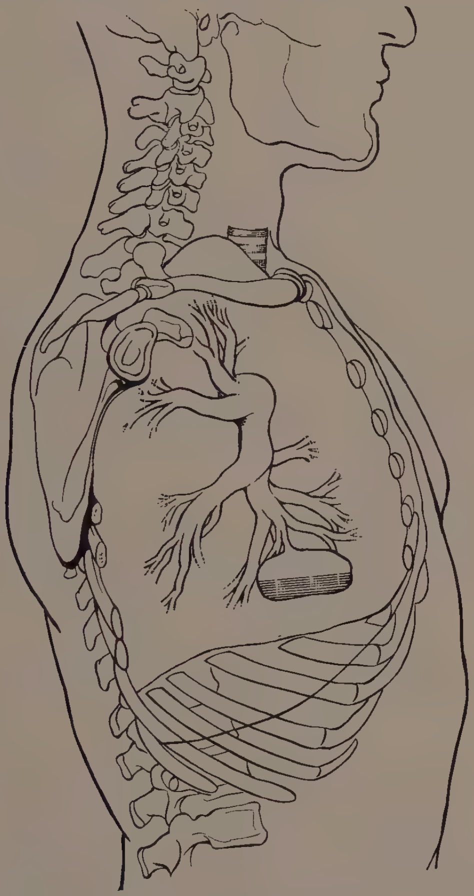


Fig. 188.—Illustrating the physical basis of Wintrich's interrupted change of sound.

The accepted physical basis for this change in pitch is, in brief, that the buccal cavity, which acts as a resonating chamber for the vibrations which are conducted to it from the pulmonary cavity by way of the respiratory passages, is tuned for different vibrations accordingly as the lips are opened or as they are closed; that is to say, that with the lips closed the buccal cavity acts as a resonator for the waves of longer amplitude and lower rate of vibration, whereas with the lips parted the buccal cavity is adapted best to the waves of less extensive amplitude and more rapid vibration, which it magnifies to the exclusion of the slower vibrations of greater amplitude.

In the elicitation of the change of sound of Wintrich the patient should occupy the seated position, as Baümler has shown that during examination in the dorsal decubitus the root of the tongue often tends to close more or less completely the orifice of the larynx and to prevent the change of pitch of the percussion sound upon opening the lips. Also the change of sound should be elicited during the same phase of the respiratory cycle, as the chink of the glottis is subject to changes in diameter during the two respiratory acts. Moreover, the act of deglutition, by occluding the laryngeal orifice effectually masks the change of sound.

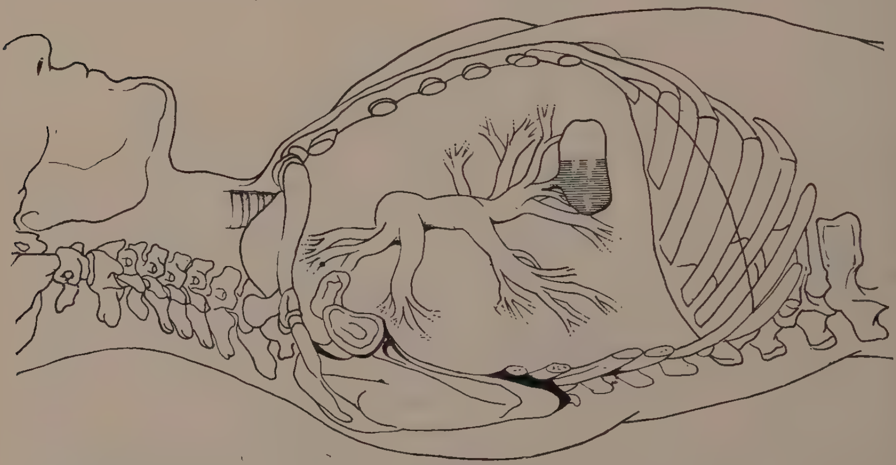


Fig. 189.—Illustrating the physical basis of Wintrich's interrupted change of sound.

WINTRICH'S INTERRUPTED CHANGE OF SOUND

When a pulmonary cavity contains fluid which is freely mobile and shifts its position readily with change in the posture of the patient, alternately occluding and leaving unoccluded the bronchial communication of the cavity, there is frequently witnessed the alternate presence and absence of Wintrich's change of sound upon changes in the attitude of the patient. When the posture of the patient is such that the bronchial communication is situated above the level of the fluid, Wintrich's change of sound is demonstrable upon percussion; whereas with the shifting of the fluid and occlusion of the bronchus with change of posture the change of sound is abolished.

When this sign can be elicited, it is of service in determining whether the bronchial communication of the cavity is situated upon its base, or upon the anterior, posterior, or lateral wall of the cavity. If the bronchus communicates with the base of the cavity, with the patient in the upright attitude the fluid occupies the base of the cavity and effectually prevents the change of sound of Wintrich. Upon placing the patient in the dorsal decubitus, the mobile fluid occupies the posterior portion of the cavity and the sign of Wintrich reappears. If the bronchial orifice is situated on the posterior wall of the cavity, the opposite condition of affairs obtains, and the change of note is demonstrable in the erect position and is abolished in the dorsal decubitus. If, on the other hand, the change of sound is demonstrable in both the erect posture and in the dorsal decubitus, but is abolished upon placing the patient in the genupectoral position, the bronchial orifice is evidently situated upon the anterior wall of the cavity. If, finally, the change of sound persists in all three of the above-named attitudes, then the bronchial orifice is situated upon one of the lateral walls of the cavity, and the change of note is obliterated upon placing the patient upon the side of the orifice.

FRIEDREICH'S RESPIRATORY CHANGE OF SOUND

Friedreich noted that the tympany which is elicited upon percussion of a superficial pulmonary cavity with patent bronchial communication possesses a higher pitch during inspiration than during expiration. This change of pitch is explained upon the basis of the increased diameter of the chink of the glottis during inspiration, the glottic slit in this instance, in continuous communication through the bronchial system with the pulmonary cavity, acting as the true orifice of the cavity and its breadth determining the pitch of the percussion tympany during the two phases of respiration. Moreover, the increased tension of the walls of the cavity during inspiration causes a slight increase in the pitch of the percussion note, which at the same time loses a portion of its tympanitic quality. Of definite diagnostic value when present, this sign is rather difficult to elicit in most cases.

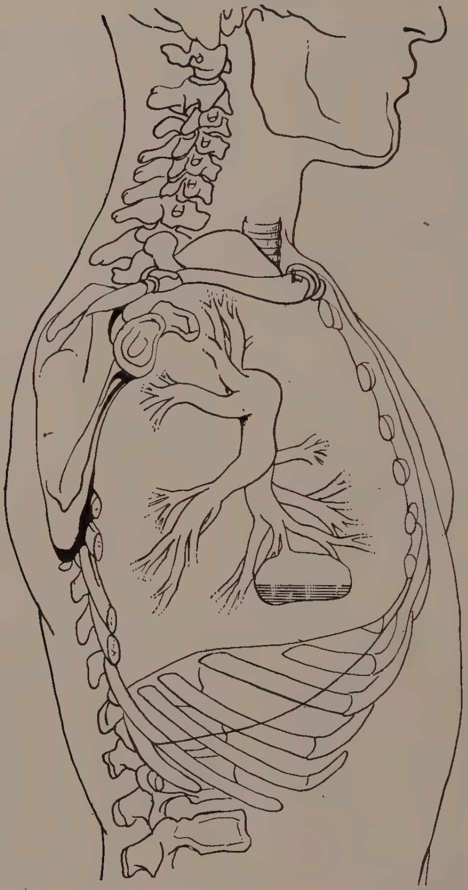


Fig. 190.—Illustrating the physical basis of Gerhardt's change of sound.

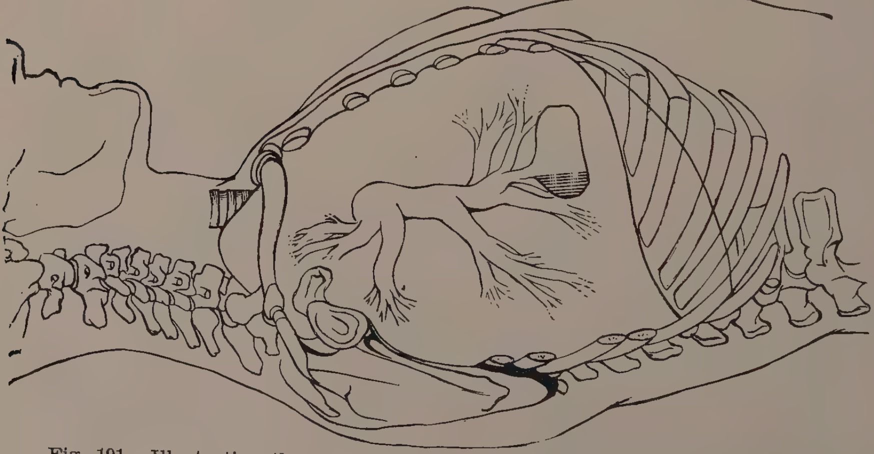


Fig. 191.—Illustrating the physical basis of Gerhardt's change of sound.

GERHARDT'S CHANGE OF SOUND

When a superficial pulmonary cavity is partially filled with mobile fluid, and when its vertical and horizontal diameters are unequal, there is noted a definite change in the pitch of the percussion note with changes in the posture of the patient. When the subject is so placed that the greater diameter of the cavity is horizontal, the tympany elicited upon percussion is of distinctly lower pitch than it is when the greater diameter of the cavity is vertical, as the result of variations in the tension of the walls of the cavity. Thus, it is observed that this sign may be utilized in the case of closed cavities in the estimation of the direction of the greater diameter of the cavity; but, as in the case of open cavities similar changes of note are observed with changes of posture, it is essential in the first place to determine through the signs which have been described whether or not the cavity is closed.

BIERMER'S PHENOMENON

Biermer noted a change of sound in the presence of hydropneumothorax analogous to the change of sound of Gerhardt; namely, that the tympanitic note elicited upon percussion over a hydropneumothorax was of lower pitch when the patient was placed in the recumbent posture, and changed to a note of higher pitch when the patient assumed the sitting posture. The variation in this instance is attributable to the change in the relative diameters of the air-containing cavity, as a result of gravitation of the fluid to the most dependent portion of the sac.

SKODAIC RESONANCE

Skodaic resonance is a hyperresonant sound which is elicited upon percussion of a portion of the lung which is in a state of relaxation or of compression. This variety of hyperresonance may be elicited in the presence of disease of the bronchi, alveolar disease, or in disease of organs adjacent to the lungs, as the pleura, pericardium, heart, or the abdominal viscera. Skodaic resonance is differentiated from tympany due to pulmonary excavation by the fact that the pitch of the sound is uniform and is not altered upon opening or closing the mouth or upon changes of posture.

In respect to the relative frequency of the etiologic factors which are concerned in the generation of Skodaic resonance, pulmonary compression stands first and foremost, and is followed by alveolar disease, and by bronchial affections.

In the presence of extensive pleural effusion the lung is elevated and is crowded into the superior portion of the pleural cavity, leading to compression and relaxation of the alveoli of the upper lobes. Hence, in this disease the examiner frequently encounters Skodaic resonance upon percussion of the thorax above the level of the fluid in these conditions. In the case of extensive pericardial effusion pressure is exerted upon the mediastinal surface of the upper lobe of the left lung, leading to compression and relaxation of this portion of the organ and the production of Skodaic resonance upon percussion of the first and second left intercostal spaces. Upon the posterior surface of the thorax, on the contrary, below the angle of the left scapula, the examiner commonly encounters dullness upon percussion, associated with bronchovesicular or bronchial breathing and increased vocal fremitus, due to a compression of the inferior lobe of the left lung to such a degree that the air is in the main forced from the alveoli, constituting Bamberger's sign of this disease. Immense cardiac hypertrophy is not infrequently attended by Skodaic resonance, which is distributed along the superior and left lateral boundaries of the area of relative cardiac dullness. Occasionally, though by no means frequently, Skodaic resonance is elicited over the superior portion of the thorax in the presence of upward displacement of the diaphragm by excessive intraabdominal pressure due to ascites, tympanites, or large abdominal tumor.

Skodaic resonance is present in the case of certain affections of the pulmonary alveoli, when the latter contain simultaneously fluid and semisolid materials. These conditions are met during the first and the third stages of lobar pneumonia, during catarrhal pneumonia, during pulmonary edema, and in pulmonary infarction of extensive distribution. The presence of dullness over both bases posteriorly with Skodaic resonance over the superior portions of the thorax anteriorly is very suggestive of catarrhal pneumonia. Hutinel and Paisseau hold that Skodaic resonance in the infraclavicular regions is the first intimation of the presence of infantile catarrhal pneumonia, making its advent before the development of impaired resonance is manifested over the bases posteriorly.

When, in the development of acquired atelectasis due to bronchial obstruction, the obstruction becomes complete, the air which is contained in the alveoli distal to the bronchial occlusion is gradually absorbed, and with the consequent relaxation of the pulmonary tissues, Skodaic resonance makes its appearance.

WILLIAMS' TRACHEAL TONE

Upon percussion of the apices of the lungs in the supraclavicular and infraclavicular regions, in the presence of apical consolidation the percussion sound frequently has imparted to it the tympany of the adjacent trachea, constituting the tracheal tone of Williams. As the fundamental cause of the sound is the vibration which is generated in the aerial content of the trachea, the tympany of Williams is subject to the variation in pitch of Wintrich upon opening and closing the mouth. In the elicitation of the note, forcible percussion is employed over the supraclavicular and infraclavicular regions.

AMPHORIC RESONANCE

Amphoric resonance is a tympanitic note of clanging, echoing, metallic quality, and of decidedly longer duration than pure tympany. A similar resounding note is elicited upon striking the side of a barrel which is empty or partially filled with water, or upon speaking in a loud voice in an empty chamber with high ceiling or in a vaulted cellar.

Amphoric resonance is generated in percussion of superficial pulmonary cavities of irregular configuration, and with smooth, tense walls, which are free from secretion. The sound is elicited over both open and closed cavities, and the pitch of the note varies with the vertical diameter of the cavity. Wintrich explained the generation of the amphoric sound on the assumption of the creation during percussion of vibrations in the air content of varying amplitude, the more rapid overtones dying away more quickly than does the fundamental tone of greater amplitude, producing an echoing sound of considerable duration.

The intensity of the amphoric percussion sound is influenced by the thickness of the walls of the cavity, by the distance of the cavity from the thoracic wall, by the thickness of the thoracic wall, and by the force of the percussion blow. In the case of open cavities, the intensity is increased when the mouth is opened.

THE CRACKED-POT SOUND (BRUIT DE POT FÊLÉ; MONEY-CHINK RESONANCE)

The cracked-pot sound is a variety of tympany in which the percussion note bears some resemblance to the sound which is produced upon tapping the side of a cracked metal jar, or to the muffled chink of coins, whence the name money-chink resonance. Neither of the above comparisons gives a true conception of the quality of the sound, which is hissing rather than metallic in quality; and its quality is more aptly imitated by the sound which ensues upon suddenly striking the clasped palms upon the knee with the sudden escape of the air confined between the palms.

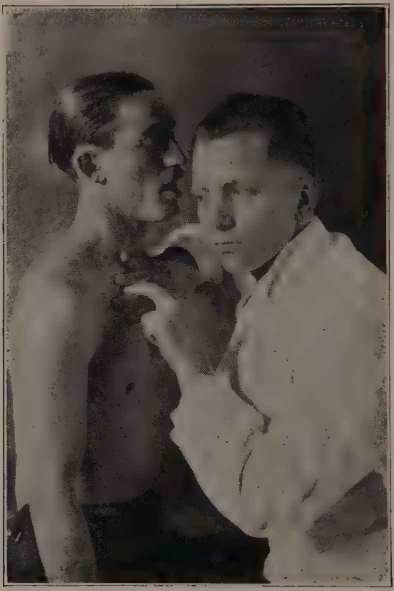


Fig. 192.—Elicitation of cracked-pot sound.

The essential element in the production of the cracked-pot sound is the sudden expulsion of air from a portion of the lung or pleural cavity, whether it be from a pulmonary cavity with free bronchial communication, from a relaxed portion of the lung, or from a pneumothorax with communicating bronchial fistula. Hence, the sound bears a variable significance, depending upon the concomitant signs in the given case. In infants and young children a cracked-pot sound possesses little significance, as it is frequently a normal phenomenon upon percussion of the thin, resilient chests of this class of subjects.

In the elicitation of the cracked-pot sound the percussion blows should be delivered slowly and forcibly during expiration, with the subject's lips parted, the examiner's ear meanwhile being held near the lips of the patient.

In the case of pulmonary cavities the sound is only elicited in cavities possessing a patent bronchial communication, whence it follows that the cracked-pot sound is attended by the change of sound of Wintrich. In the event that the cavity contains fluid which, upon change of posture, causes interruption of the change of sound of Wintrich, the cracked-pot sound also disappears. A further essential for the elicitation of the cracked-pot sound from pulmonary cavities is that the cavity be situated in the periphery of the lung and that the thoracic parietes be sufficiently thin and elastic to permit the expulsion of the air from the cavity during the delivery of the percussion blow. Grancher has noted the disappearance of the cracked-pot sound over superficial cavities after a few percussion blows and its reappearance following several deep inspirations. He assumes that the air is partially expelled from the cavity during the primary percussion, and that the sound only again becomes evident upon replacement of the aerial content of the cavity during deep inspiration. In the case of a cavity containing fluid or viscid secretion, abolition of the sound may be due to bronchial occlusion.

In the presence of apical pneumonia, apical consolidation of chronic phthisis, or in compression of the superior lobe of the lung by an immense pleural effusion it is occasionally possible to obtain a cracked-pot sound upon percussion of the anterior thoracic wall in the first and second intercostal spaces. In this case the cracked-pot sound is likely to be attended by the tracheal tone of Williams.

During the first and third stages of lobar pneumonia the cracked-pot sound is occasionally demonstrable in the infraclavicular and mammary region upon the side of the disease. Cockle has observed the sign in simple catarrhal bronchitis in children; and Rollet has encountered it in bronchopneumonia.

In the case of open pneumothorax, whether the fistulous opening be external upon the surface of the thorax, or internally as a result of rupture of a tuberculous cavity of the lung, the cracked-pot sound is frequently to be elicited. Nothnagel, Oppolzer, Frerichs, and Eichhorst have encountered the sound in association with this disease.

When the cracked-pot sound is encountered at the apices, it is plainly a question of pulmonary compression or of pulmonary cavity with bronchial communication, which is probably tuberculous in origin. In cases of tuberculous origin Loeb holds that the sound is most frequently encountered over the right apex.

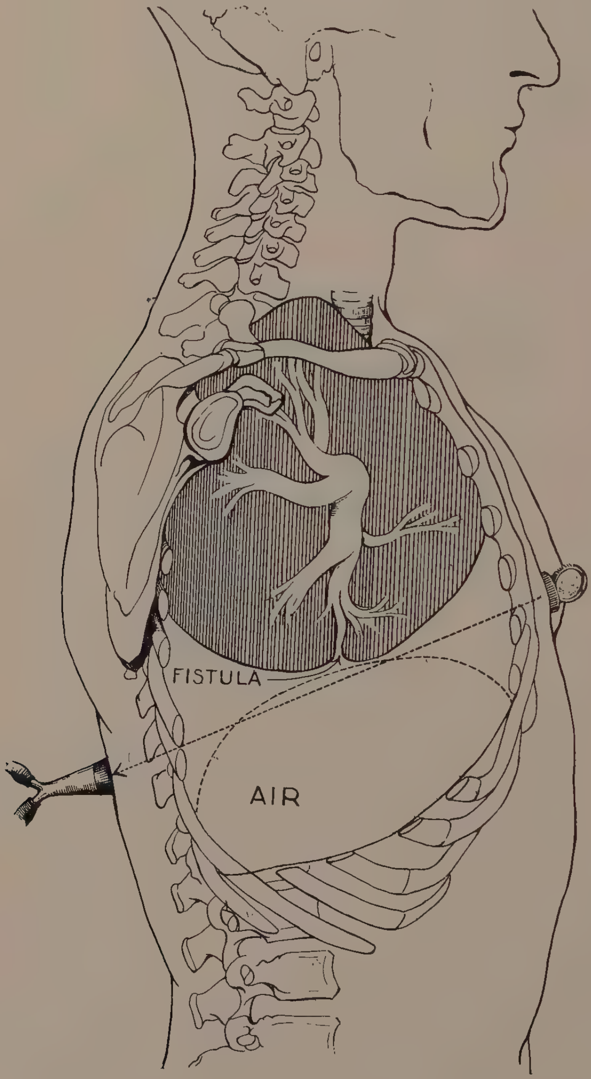


Fig. 193.—Illustrating bell tympany, or Gairdner's coin test.

Bronchiectatic dilatations of the bronchi furnish all of the physical requirements for the generation of the cracked-pot sound, the chink of the glottis in this instance playing the rôle of the constricted orifice through which the air is expelled during percussion. However, bronchiectases are usually too deeply seated in the pulmonary parenchyma to be materially influenced by the force of the percussion blow, and, moreover, bronchiectasis is not a disease which is commonly encountered.

GAIRDNER'S COIN TEST (BELL TYMPANY; ANVIL TEST)

In cases of pneumothorax, when the stethoscope is applied to the base of the thorax posteriorly while an assistant percusses the anterior surface of the thorax with two coins employed as pleximeter and plexor, respectively, an echoing, metallic sound is appreciated through the stethoscope, possessing a ringing quality analogous to the distant ring of a hammer upon an anvil.

The coin test can, likewise, occasionally be elicited over a large superficial pulmonary cavity, which is empty and the walls of which are in a state of partial relaxation. The sign is much more constant, however, in cases of pneumothorax, of which it constitutes a very reliable sign.

CHAPTER XXIV

PERCUSSION OF THE HEART

Percussion is employed in the study of the cardiovascular system principally in the determination of the position of the heart, in the detection of alterations in its shape and size, and in the detection of the presence of fluid in the pericardial sac. Percussion is likewise employed to advantage in estimating the dimensions of the area of dullness produced by the great vessels springing from the base of the heart in the area of vascular dullness.

The size, shape and position of the heart within the thoracic cavity are determined by outlining upon the surface of the thorax the areas of relative and absolute cardiac dullness.

TECHNIC OF CARDIAC PERCUSSION

The areas of relative and absolute cardiac dullness, representing roughly the size, shape and position of the heart, may be outlined by ordinary mediate percussion, threshold percussion, or auscultatory percussion.

In the practice of threshold percussion the pleximeter finger is bent at a right angle at the junction of the first and second phalanges and the tip of the finger is closely applied to the intercostal space, avoiding contact with the ribs or costal cartilages. The percussion blow is delivered lightly upon the first phalanx, thereby setting up vibrations in a very restricted area of the thorax.

Whatever method of percussion is employed, the examiner should commence to percuss from three directions in order to fix the superior and lateral borders of the heart, employing as he proceeds both deep and superficial percussion.

In fixing the right and left borders, the examiner should percuss from the right and left axillary regions upon either side of the thorax in the third, fourth and fifth interspaces toward the precordia, employing relatively deep percussion until impairment of resonance indicates that the lateral borders of the area of relative cardiac dullness have been attained. Having marked these points upon the thoracic surface, the examiner should continue the percussion along the same lines, substituting superficial percussion for the deep strokes heretofore employed until the note changes to flatness, indicating that the lateral margins of the area of absolute cardiac dullness have been reached, representing the portion of the right ventricle which is in direct apposition with the anterior thoracic wall.

After having marked these several points upon the surface of the thorax, the examiner, beginning in the left infraclavicular region, should percuss downward along the interval between the left sternal and parasternal lines, employing in the first instance deep percussion; and, when the superior limit of the area of relative dullness has been attained, he should continue with superficial percussion until the upper limit of the area of absolute cardiac dullness is reached, which is marked upon the surface of the thorax.

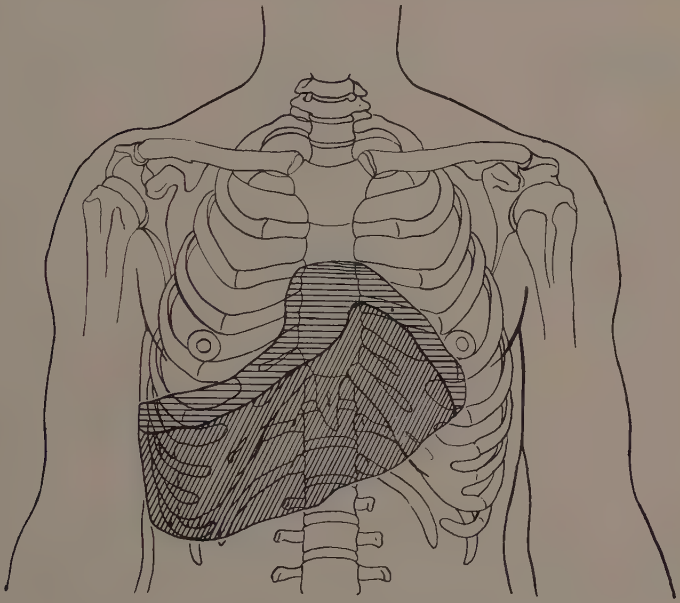


Fig. 194.—Areas of cardiac and hepatic dullness and flatness.

By connecting the points upon the chest wall at which the initial change in the percussion note was observed in each instance, the areas of relative and absolute cardiac dullness are graphically represented upon the surface of the thorax.

The delimitation of the dullness of the heart by percussion affords data sufficiently accurate for routine clinical work; but the cardiac outline thus obtained never represents the exact size and position of the heart. As a rule, the outline obtained errs one to two centimeters to the left of the right lateral border, while there is usually a like error toward the left in delimitation of the left lateral border of the heart. Over the superior sternal region it is not possible to distinguish between the areas of cardiac and vascular dullness by percussion.

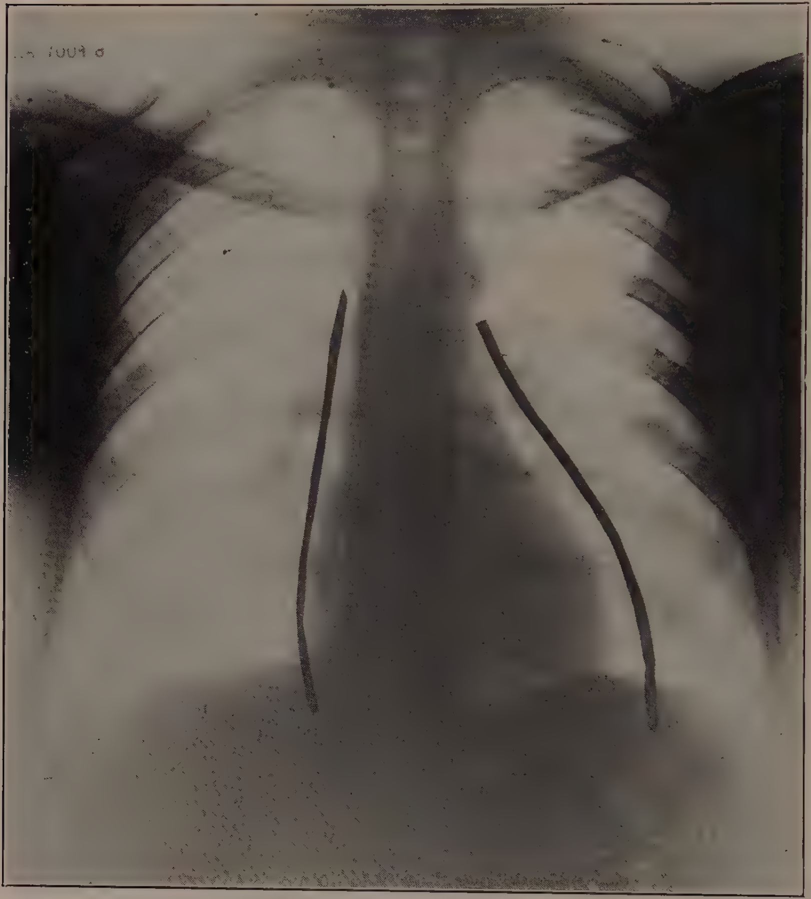


Fig. 195.—Radiograph of the chest taken with x-ray tube 6 feet from the subject. Dark lines are made by placing lead strips over the area of heart dullness as outlined by percussion. Note discrepancy between the radiographic and percussion outline.

Due to change in distance between patient and x-ray tube the cardiac outline on fluroscopic examination more nearly corresponds to the percussion outline.

In the practice of cardiac percussion, the examiner should utilize as far as possible the information furnished by the progressive increase of resistance which is encountered as the cardiac borders are approached. Piorry, Traube and Ebstein have each in turn emphasized the importance of this factor; and, indeed, Ebstein developed a special method of delimitation of the heart based upon the information thus obtained.

When for any reason the findings upon percussion of the precordia are obscure, resort should be had to fluoroscopy and radiography of the heart.

Covering the percussion outline of the heart, determined in the upright position, with narrow strips of lead, and placing the subject before the fluoroscope, gives a fair check on the accuracy of percussion. The distortion of the fluoroscopic outline varies with the distance of the subject from the x-ray tube, but on the average will be found to be remarkably close to that of the cardiac outline as percussed by an experienced clinician.

The x-ray film taken at six feet usually shows the cardiac shadow slightly smaller than the percussion outline, as the lead lines are farther from the tube than the heart. Films taken at a shorter distance show less variation.

AREAS OF CARDIAC DULLNESS

When the examiner percusses toward the heart from various points in its vicinity upon the surface of the thorax, two changes in the quality and pitch of the percussion note are observed. As the borders of the heart which are covered by the anterior borders of the lungs are approached, the normal vesicular resonance becomes impaired; and, finally, when the portion of the right ventricle which lies in direct apposition with the chest wall is reached, the note becomes frankly flat. In this manner there may be defined upon the anterior surface of the thorax two areas of cardiac dullness, the one within the other. The inner area, representing the region in which the heart is uncovered by the anterior borders of the lungs, is termed the *area of absolute cardiac dullness,* or the *area of cardiac flatness;* whereas, the peripheral area, representing the portion of the heart which is overlapped by the anterior pulmonary borders, is termed the *area of relative cardiac dullness.*

The *area of absolute cardiac dullness,* representing the limited portion of the right ventricle which is in direct apposition with the anterior thoracic wall, corresponds to a roughly triangular area, which is bounded upon the right by a vertical line drawn along the left border of the sternum from the level of the fourth costal cartilage to the upper border of the sixth costal cartilage, and upon the left by a line drawn downward and outward from the junction of the fourth left costal cartilage with the sternum to the upper border of the sixth rib in the left parasternal line, corresponding to a point approximately one-half inch internal to the cardiac apex. Inferiorly the area of absolute cardiac dullness is continuous with the area of flatness of the liver.

The *area of relative cardiac dullness,* representing the portion of the heart which is overlapped by the anterior borders of the lungs, is bounded upon the right by a vertical line drawn upon the chest wall from the upper border of the third costal cartilage near its junction with the sternum along the right sternal border to the upper border of the sixth costal cartilage, and upon the left by a slightly curved line with its convexity directed toward the left and upward, drawn from the third left chondrosternal junction to the fifth intercostal space one-half inch internal to the midclavicular line. Inferiorly the area of relative cardiac dullness blends with the area of relative hepatic dullness.

Thus, the areas of cardiac dullness roughly represent a triangle within a triangle, the area of absolute cardiac dullness lying within the confines of the area of relative dullness of the heart save inferiorly, where the boundaries are the same, and where the flatness of the heart blends imperceptibly with that of the liver. Similarly, the right border of the area of relative cardiac dullness joins the superior border of the area of hepatic dullness at almost a right angle, the angle of pulmonary resonance in the fifth right interspace formed in this manner constituting *Ebstein's cardiohepatic angle.*

VARIATIONS IN THE AREAS OF CARDIAC DULLNESS

The areas of cardiac dullness present certain variations in position and outline depending upon the age of the subject, the posture assumed by the patient, the contour of the thorax, the position of the diaphragm, and the presence or absence of disease of the heart, pericardium or adjacent viscera.

During childhood the area of relative cardiac dullness is more extensive than at any later period of life. Owing to the greater flexibility of the thorax and the great elasticity of the lungs obtaining at this time of life, a relatively greater portion of the heart is exposed to the thoracic wall. Moreover, in the child the heart occupies a higher position in the thoracic cavity than is the case in the adult subject.

In children it is not uncommon to find the superior limit of the area of cardiac dullness occupying the second interspace in the left parasternal line, the left border extending to the midclavicular line in the third interspace, and surpassing this line by one-eighth to one-half inch in the fourth interspace, the point corresponding to the apex of the heart in this class of subject. Similarly, during childhood the area of absolute cardiac dullness is increased in a transverse direction, and is raised to the extent of one intercostal space.

In the aged subject, on the contrary, with the physiologic ptosis of the heart and abdominal viscera obtaining at this time of life, the areas of cardiac dullness occupy a lower level, the superior limit approximating the lower border of the fourth rib in the left parasternal line, while the left border approaches more nearly the left sternal border.

The dimensions of the areas of cardiac dullness vary with the posture assumed by the patient. In the upright posture the heart is in relation with a more extensive portion of the anterior thoracic wall, and the area of dullness as elicited upon percussion is extended to a corresponding degree. Upon assuming the right lateral decubitus, the area of dullness is displaced toward the right side of the thorax. Under these circumstances the area of cardiac flatness adjacent to the left sternal border is diminished or disappears entirely, while the area of dullness is distinctly extended to the right of the sternum. Upon assuming the left lateral decubitus, the area of cardiac flatness is increased transversely, and the area of cardiac dullness is carried approximately to the left midclavicular line.

In the robust subject with a deep thorax the areas of cardiac dullness are diminished in extent as outlined by percussion; whereas, in the case of the flat expiratory type of thorax in the normal subject, and in the presence of deficient inflation of the anterior pulmonary borders, the areas exhibit a corresponding increase in extent.

Depression of the diaphragm incident to advancing age or increased intrathoracic pressure is attended by a descent of the areas of cardiac dullness, while upward displacement of the diaphragm incident to increased subphrenic pressure is attended by elevation of the areas of dullness of the heart.

In the presence of disease of the heart, pericardium or other thoracic viscera the areas of cardiac dullness may exhibit a general increase or diminution in their outlines, or alterations of the outline in certain directions only may be encountered.

General Increase.—A general increase in the area of cardiac dullness in all directions is indicative of cardiac hypertrophy or dilatation, pericarditis with effusion, or a tumor of the mediastinal structures which pushes the heart forward. In pericarditis with effusion the area of dullness is roughly triangular or pear-shaped, with the base directed downward toward the diaphragm.

General Decrease.—Retraction of all the borders of the area of cardiac dullness is indicative of cardiac atrophy or of pericardial adhesions which draw the heart under the anterior pulmonary borders. Hypertrophic emphysema, by interposing the voluminous anterior borders of the lungs between the heart and chest wall, causes a general decrease in the area of cardiac dullness in the absence of cardiac pathology.

Displacement of the area of cardiac dullness, as indicated by displacement of the cardiac impulse, occurs in extensive pleurisy with effusion, the heart being displaced toward the side of the thorax opposite to the effusion. The traction of postpleuritic adhesions tends to draw the heart toward the side of the diseased pleura, with consequent shifting of the area of cardiac dullness. Subphrenic pressure in ascites, tympanites, hepatic enlargement or abscess, causes displacement of the area of cardiac dullness upward and toward the left side of the thorax. In all of these conditions the entire area of cardiac dullness is displaced; but, in the absence of coincident disease of the heart or pericardium, it is of normal dimensions.

Upward Increase.—Extension of the area of cardiac dullness in an upward direction accompanies pericarditis with effusion, and in the presence of aneurysm of the ascending portion or arch of the aorta a similar extension upward is observed. In pericarditis with effusion the area becomes irregularly triangular in outline, with the base directed downward as a result of the characteristic configuration of the pericardial sac.

Increase to the Left.—An increase in the area of cardiac dullness toward the left occurs with hypertrophy and dilatation of the left ventricle, and in cardiac displacement by mediastinal

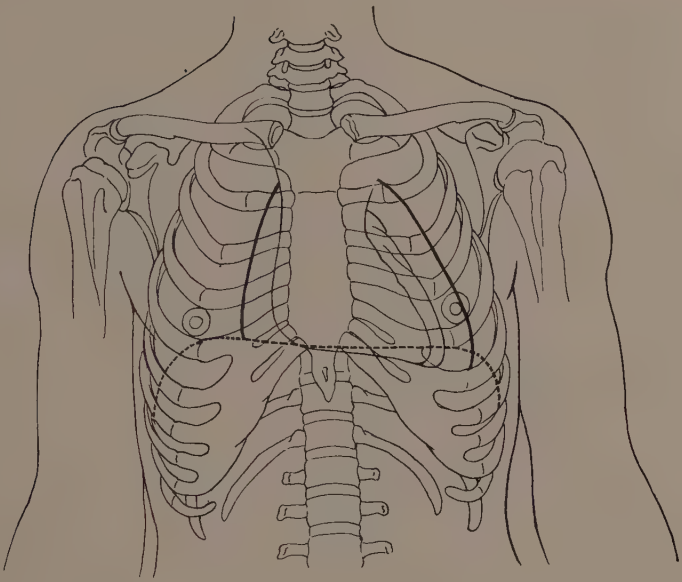


Fig. 196.—General extension of cardiac dullness in extensive pericardial effusion.

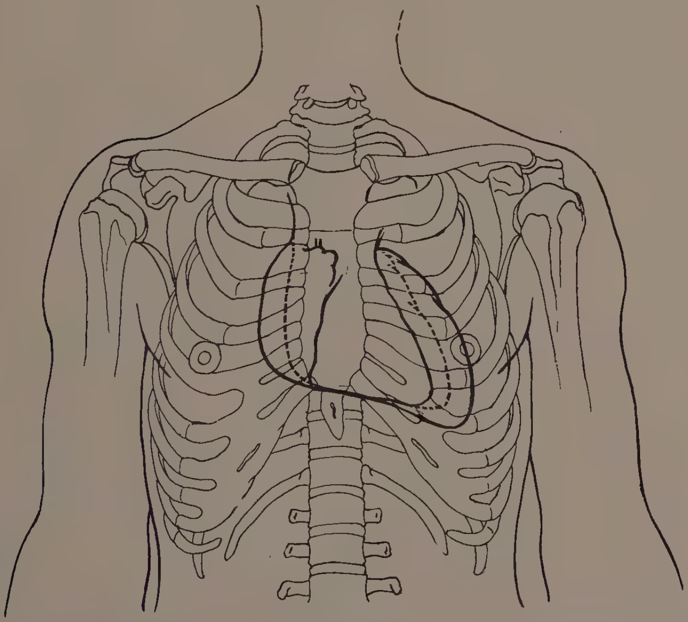


Fig. 197.—Extension of cardiac dullness toward the right and toward the left and downward in combined right and left ventricular hypertrophy. The dotted lines indicate the normal cardiac outline.

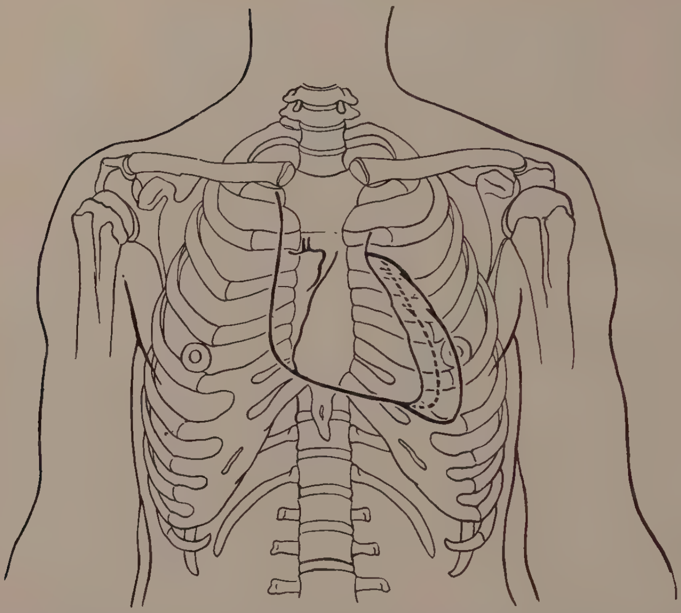


Fig. 198.—Extension of cardiac dullness toward the left and downward in left ventricular hypertrophy. Dotted lines indicate normal cardiac outline.

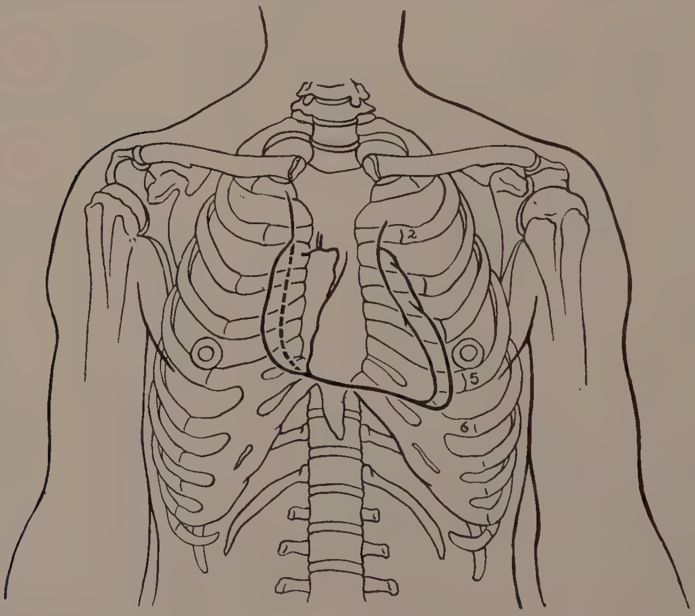


Fig. 199.—Extension of cardiac dullness toward the right in right ventricular hypertrophy. Dotted lines indicate normal cardiac outlines.

pressure. In left ventricular hypertrophy the area is increased downward as well as toward the left, occasionally reaching the seventh interspace in the anterior axillary line.

Increase to the Right.—Extension of the area of cardiac dullness toward the right, the dullness of the heart encroaching upon the normal vesicular resonance of Ebstein's cardiohepatic angle, occurs with right ventricular and auricular hypertrophy and dilatation, and in pericarditis with effusion, constituting in the latter disease *Rotch's sign.* A distended inferior vena cava may be responsible for a slight extension of the area of dullness to the right of the sternum.

AREA OF VASCULAR DULLNESS

The area of vascular dullness, overlying the great vessels springing from the base of the heart, occupies the sternal region from the episternal notch to the level of the third costal cartilage. In the lower portion of this region the dullness of this area blends imperceptibly with the superior limit of the area of cardiac dullness.

An extension of this area of dullness toward the right in the first and second intercostal spaces occurs with aneurysm of the ascending aorta.

CHAPTER XXV

PERCUSSION OF THE ABDOMEN

In the examination of the abdomen the examiner may employ ordinary mediate finger percussion; he may resort to flicking percussion; or he may employ auscultatory percussion. In the determination of minor grades of impairment of intestinal or gastric tympany flicking percussion is very serviceable, while in outlining the various solid viscera and tumors of the abdominal organs auscultatory percussion is a very serviceable and dependable method of procedure.

In the practice of flicking percussion, the middle finger of the left hand should be applied upon the abdominal surface with the nail downward, while the middle finger of the right hand is so flexed that the nail of the finger is pressed firmly against the palmar aspect of the thumb. The percussion stroke is delivered by permitting the right middle finger to escape in such manner as to strike firmly and quickly against the palmar aspect of the finger applied to the abdominal surface.

In the practice of mediate percussion of the abdomen the pleximeter finger is in the first instance applied lightly to the integument, and the blow is delivered upon the terminal phalanx. As the percussion proceeds the pleximeter should be gradually depressed or sunk into the abdominal wall, thereby gaining closer proximity to solid organs or masses within the abdomen.

In general, the percussion note is tympanitic upon percussion of the hollow intraabdominal viscera when these contain a moderate quantity of gas unmixed with any excess of solid material, changing to a flat note over the solid intraabdominal organs. A similar flat note is elicited over solid intraabdominal tumors and over collections of fluid, whether encysted or free in the peritoneal cavity.

During percussion of the abdomen the patient should assume the dorsal decubitus with the abdominal wall freely exposed to the pubes. In delimiting the borders of solid organs and tumors, and in the estimation of the relative tympanicity of the adjacent hollow viscera auscultatory percussion, with the employment of a superficial percussion blow or a light stroking movement upon the abdominal surface, is most serviceable. The technic of aus-

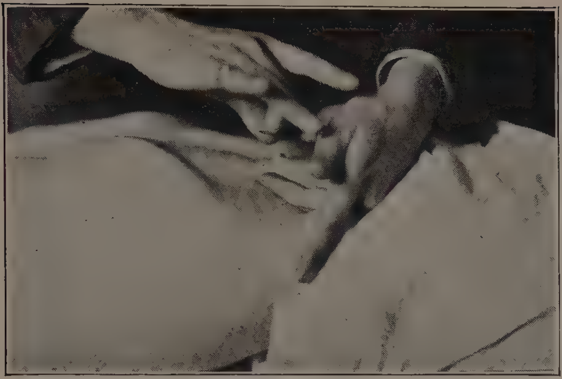


Fig. 200.—Flicking percussion of abdomen.

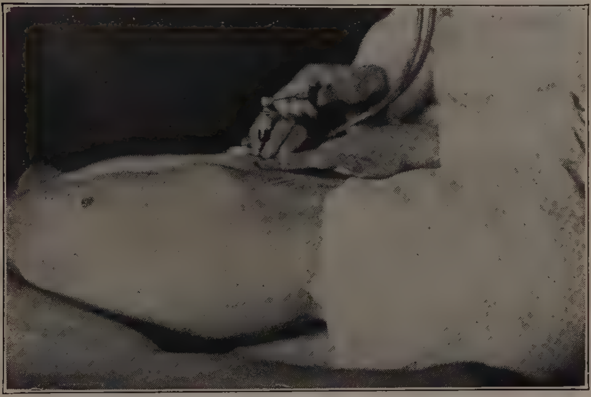


Fig. 201.—Auscultatory percussion of stomach.

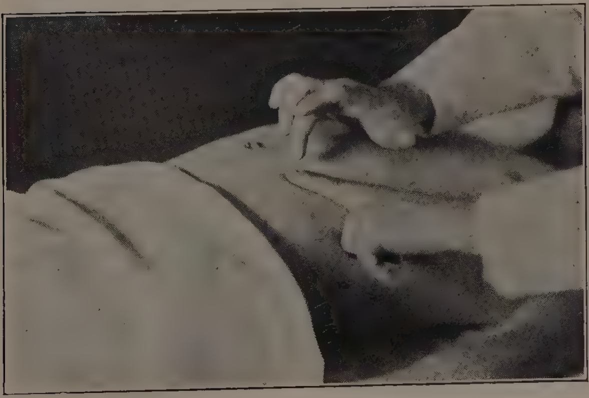


Fig. 202.—Percussion of abdomen. In first step finger is applied lightly to integument.

cultatory percussion as applied to the examination of the abdomen differs in nowise from the technic which has been described in a previous section.

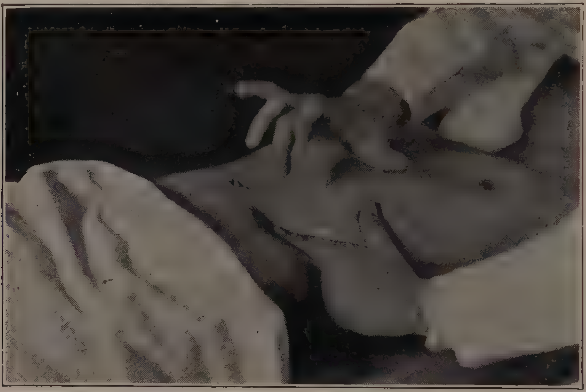


Fig. 203.—Percussion of abdomen. In second step the finger is pressed firmly, so as to thin out the wall and get closer to deep structures.

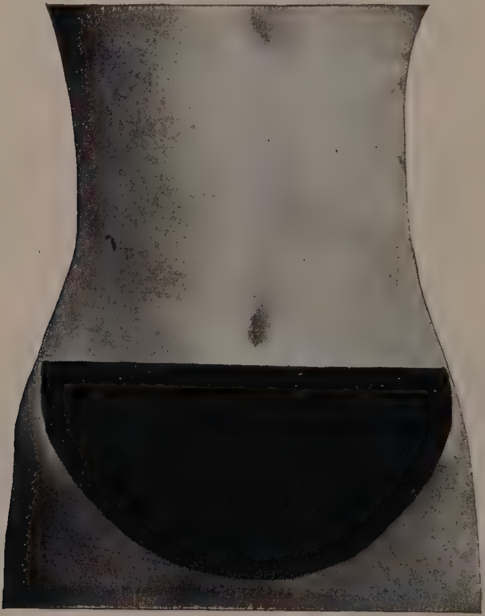


Fig. 204.—Indicating the area of dullness in moderate ascites, with the patient standing. (From Crossen.)

The details of technic of outlining the various abdominal viscera are discussed in their appropriate sections.

THE LIVER

In practicing percussion of the hepatic area the student succeeds in establishing two gradations of dullness due to the presence of the liver in the upper and right segment of the abdomen below the lower ribs and intercostal spaces. These areas constitute respectively the areas of hepatic dullness and of hepatic flatness.

The Areas of Hepatic Dullness and Flatness.—Upon percussing downward over the surface of the thorax and abdomen in the midclavicular, midaxillary, and scapular lines from an area of frank vesicular resonance, the percussion note will become impaired or dull when the point is reached where the superior limit of the liver is covered by the pulmonary tissues. The point of change in the quality of the note indicates the upper limits of the *area of hepatic dullness.* The superior limits of this area are encountered at the upper border of the fifth rib in the right midclavicular line, the seventh intercostal space in the right midaxillary line, and at the eighth intercostal space in the right scapular line.

Upon continuing the percussion downward along these lines, substituting light for forcible percussion, a point is reached in which the dullness gives place to flatness, indicating the upper limit of the region where the liver is in direct contact with the abdominal wall, the superior limit of the *area of hepatic flatness.* The superior limits of this area are found in the normal adult subject at the lower border of the sixth rib in the right midclavicular line, of the eighth rib in the right midaxillary line, and of the tenth rib in the right scapular line.

If now the percussion is continued downward along the same lines, the flat note will be replaced by intestinal tympany when the inferior limit of the liver is attained. The points of change indicating the inferior limit of the area of hepatic flatness are encountered at the ninth rib in the right midclavicular line, the tenth rib in the right midaxillary line, while in the right scapular line the flatness of the liver is continuous with that produced by the kidney.

The inferior limit of the area of hepatic flatness in the epigastric region lies three to four inches below the ensiform cartilage. Thus, it is observed that the areas of hepatic dullness and flatness extend downward and toward the right; that posteriorly they are continuous with the flatness of the right kidney; and

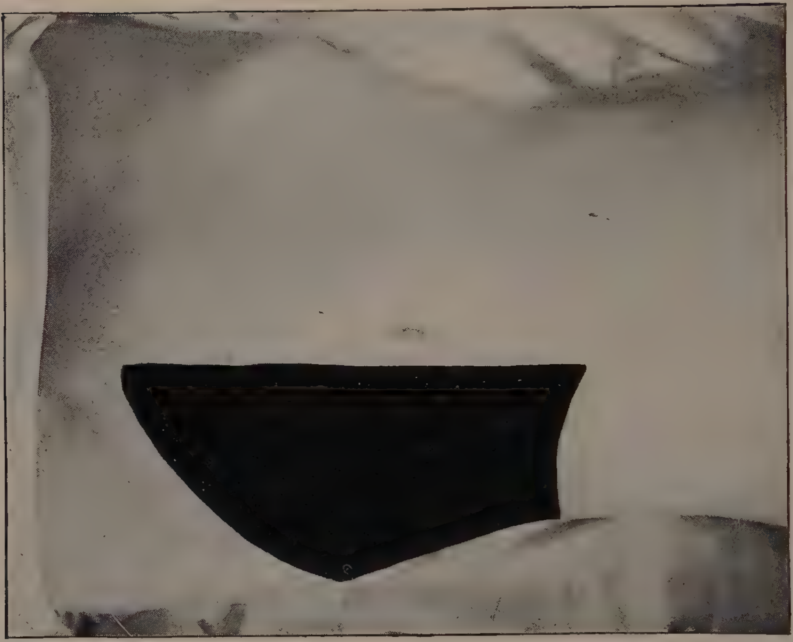


Fig. 205.—Indicating the area of dullness in a case of moderate ascites, with the patient turned on the left side. (From Crossen.)

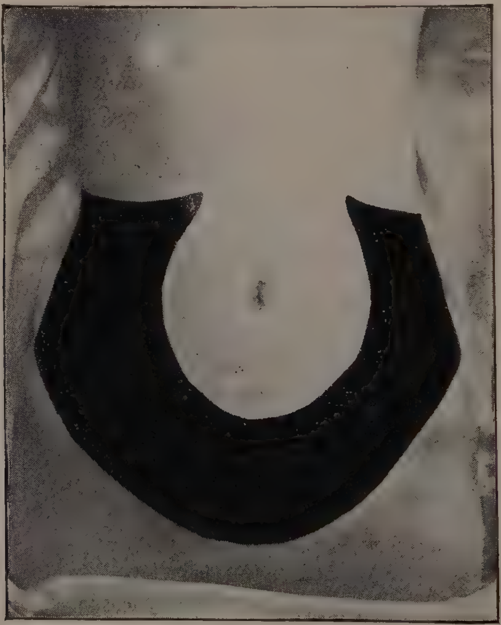


Fig. 206.—Showing the area of dullness in moderate ascites, with the patient lying on her back. (From Crossen.)

that anteriorly they blend with the right border of the area of cardiac dullness.

In pathologic states the hepatic dullness may be abolished, may be diminished in extent, may be increased in extent, or the area of dullness as a whole may be displaced from its normal site.

Absence of hepatic dullness upon percussion of the hepatic area is significant of falling of the liver, in which event the dullness of this organ is replaced by intestinal tympany. This state, which is usually encountered in multiparæ as a result of extreme relaxation of the suspensory ligaments of the liver, may permit the liver to occupy the iliac fossa, constituting the ''floating liver'' of Cantani. In this abnormal situation the liver could readily be confused with an abdominal tumor. In this connection it is to be remembered that the displaced liver occupies in the main the right half of the abdominal cavity; that its superior surface is convex, smooth, and rounded; and that palpation of its inferior sharp margin reveals the presence of the two notches which exist upon this border of the organ. The displaced liver can usually be restored to its normal habitat by manual manipulation, whereupon the intestinal tympany is replaced by hepatic dullness in that region.

When tympanites is present in a pronounced degree, the liver is not infrequently crowded upward into the right vault of the diaphragm and percussion of the hepatic area under these circumstances yields only mediocre dullness in comparison with the excessive tympanicity of the percussion note over adjacent portions of the abdomen. Similarly when the liver and inferior diaphragmatic surface have contracted adhesions which exert traction upon the superior surface of the organ, the area of hepatic dullness is very much restricted and occasionally is outlined with difficulty if indeed at all. Also in transposition of the viscera, when the spleen occupies the right upper quadrant of the abdominal cavity and the liver lies in the upper left quadrant, the area of hepatic dullness is defective. As a rule in these cases the thoracic viscera are also transposed, the cardiac apex lying upon the right side of the sternum; but Mosler has encountered cases in which the transposition of the viscera was limited solely to the liver and the spleen.

Diminution of hepatic dullness is encountered in all cases of diminution in the dimensions of the liver; but it would be erroneous to immediately conclude that the liver has diminished in volume in every case in which there is a restriction of the area of hepatic dullness. Before definite conclusions may be formulated in respect to the dimensions of the liver, it is necessary to establish the absence of certain conditions which may simulate a diminution in the volume of the liver. Occasionally the large intestine insinuates itself in front of the lower border of the liver, simulating a diminution in volume of this organ. In these cases it is necessary to endeavor by forcible compression with the pleximeter to reach the subjacent hepatic surface and to obtain hepatic dullness through the tympanitic superjacent colon. Similarly, in the presence of well established hypertrophic emphysema, the lower border of the right lung is extended and comes to overlie a greater area of the anterior and lateral hepatic surfaces, thus simulating a diminution in the volume of the liver. Extreme tympanites acts similarly in masking the true level of the inferior border of the area of hepatic flatness. Similarly all conditions which are attended by increase of general intraabdominal pressure, such as ascites, a large tumor of the intestine or an ovarian cyst, by forcing the liver upward into the vault of the diaphragm cause a pseudodiminution in the volume of the liver as outlined by percussion.

Diminution of the area of hepatic dullness attributable to diminution of the volume of the liver is encountered in acute yellow atrophy, atrophic hepatic cirrhosis, and hepatic atrophy consecutive to obliteration of the biliary passages. In the case of acute yellow atrophy there is produced during the course of a few days a very notable diminution in the volume of the liver, which is contracted into a small pulpy mass adjacent to the vertebral column and covered by coils of the small intestine. In the case of atrophic hepatic cirrhosis the retraction of the gland is first noted in the left lobe, which is retracted toward the right, with the production of a gastrocardiac limit of the semilunar space of Traube, which comes to intervene between the gastropulmonary and gastrohepatic limits of this area of frank gastric tympany. This form of hepatic cirrhosis is usually attended by enlargement of the spleen and ascites. In hepatic atrophy consecutive to obliteration of the biliary passages, the diminution in volume of the liver is preceded by a transient hypertrophy of the organ, and is accompanied by chronic icterus.

Increase of the area of hepatic dullness is encountered in the presence of hypertrophic cirrhosis and in degeneration of the organ, in the presence of hepatic tumor or abscess, and in simple congestion of the organ. From the clinical standpoint it is desirable to distinguish two varieties of hepatic hypertrophy; namely, hypertrophy in which the liver preserves its normal contour, and hypertrophy of the organ which develops irregularly, with the presence of abnormal protrusions upon the hepatic surface.

A generally enlarged liver with maintenance of the normal contour is encountered in the hypertrophic cirrhosis of Hanot. A similar regular enlargement of the liver, but which is not frequently attended by jaundice, is encountered in fatty infiltration and chronic passive congestion of the liver in its earlier stages, in chronic malarial intoxication, during the acute infectious fevers, and in diabetes and amyloid disease of the liver, as well as in Banti's disease.

Irregular hypertrophy of the liver, with alteration of the normal contour of the organ in the form of multiple nodules upon the surface or a single extensive protuberance of the surface of the organ, is encountered in connection with carcinomatous infiltration of the liver, syphilis of the liver, hepatic abscess, or hydatid disease of the liver.

While increase in the volume of the liver results in extension of the areas of hepatic dullness and flatness, it is to be borne in mind that not every extension of these areas is symptomatic of hepatic enlargement. In all cases in which the inferior limit of the area of hepatic flatness is extended, the superior limit of hepatic dullness should be carefully delimited as well as the left sharp extremity of the left lobe of the organ above Traube's semilunar space. In true hepatic hypertrophy the area of hepatic dullness is extended in one or all of its dimensions. When the lower limit is established, the examiner should proceed to the delimitation of the superior limit with a view to the determination whether this limit occupies its normal level, whether it is elevated, or whether it is lowered, indicating in the last instance a depression of the organ rather than hypertrophy. In true enlargement of the liver the left extremity of the left lobe of the organ extends ordinarily well over into the left hypochondriac region, it may be to impinge against the spleen, with the consequent obliteration of the gastropulmonary limit of the semilunar space of Traube.

A pseudoextension of hepatic dullness superiorly is produced in the case of extensive effusion into the right pleural sac, in which event the continuous dullness may be confused with an extension of hepatic dullness upward, such as not infrequently attends hydatid cyst of the liver. The examiner should be circumspect in the differential diagnosis of these two states and should bear in mind the principle laid down by Frerichs; namely, that in the case of hydatid disease of the liver the superior limit of the zone of dullness presents its convexity superiorly, whereas in the event of pleurisy with effusion, the dullness presents an inferior convexity. A right-sided lobar pneumonia is quite as likely to cause confusion in the establishment of the superior limit of the hepatic dullness in any case in which hepatic enlargement under these circumstances is suspected. In the latter event the examiner should seek for bronchial respiration, râles, and increased vocal fremitus over the upper regions of the dull area.

A pseudoextension of hepatic dullness downward and toward the left may be encountered when the stomach and intestines contain solid material or when they are the seat of neoplasm. In the first instance evacuation of the gastrointestinal canal clears the confusing signs; while in the second case the examiner should resort to methodical palpation of the epigastrium and to auscultatory percussion of the various organs in the effort to differentiate the site of the growth, if any be present. Tumors of the stomach or intestine upon this examination are mingled with intestinal tympany and reveal amphoric phenomena, both of which are absent in the case of growths springing from the liver.

Finally, in any case in which it is a question of differentiation of collections situated above and below the diaphragm, fluoroscopy and radiography are recommended in the differential diagnosis.

Displacement of the Liver.—As stated in a previous paragraph, instead of being increased in extent in one or more of its dimensions, the area of hepatic dullness may be displaced in its entirety. This displacement is usually manifested in a vertical direction.

Downward displacement of the liver may be occasioned by the pressure upon the superior aspect of the diaphragm by hypertrophic emphysema, pneumothorax, right-sided pleurisy with effusion, cardiac hypertrophy, or large pericardial effusion. Similarly, the liver may be depressed by a subphrenic abscess. Finally, the liver may participate in a general visceroptosis and occupy a lower level in the abdominal cavity than is normal. In the case of the ''floating liver'' of Cantani, occurring in multiparæ, the liver may occupy the right iliac fossa.

Downward displacement of the liver is differentiated from an increase in volume of the organ by demonstrating by percussion that the superior limit of hepatic dullness occupies a lower level than is normal.

Upward displacement of the liver occurs as a result of pressure exerted upon the inferior surface of the organ by ascites, tympanites, or increased abdominal tension due to tumor of an abdominal organ or ovarian cyst. Upward displacement may, however, be the sequence of diminished intrathoracic tension such as occurs with fibroid retraction of the right lung; or it may be the result of paralysis of the diaphragm. In upward displacement of the liver it may be demonstrated by percussion that both the superior and inferior limits of hepatic dullness are elevated and not the superior limit alone.

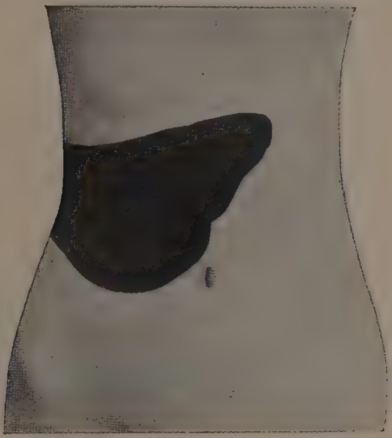


Fig. 207.—Indicating the region for dullness from enlarged liver. (From Crossen.)

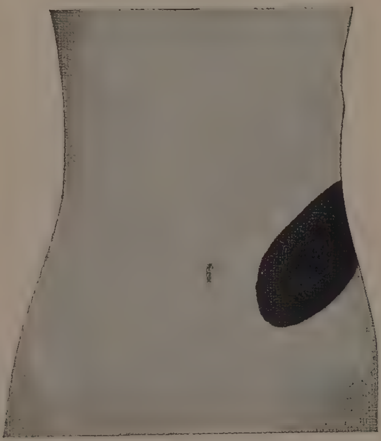


Fig. 208.—Indicating the region for dullness from enlarged spleen. (From Crossen.)

THE SPLEEN

The inherent difficulties attending delimitation of the splenic dullness by percussion cannot be too strongly emphasized. In delimiting the organ, the student may sometimes employ auscultatory percussion to better advantage than simple pleximetric percussion. Owing to the anatomical situation of the spleen between the inferior border of the left lung, the fundus of the stomach, and the summit of the left kidney, in percussion of the spleen two angles of gastric and colonic tympany are encountered. The first of these angles is encountered at the level of the ninth rib in the posterior axillary line, corresponding to the course of the superior and anterior borders of the spleen, the *splenopulmonary angle*, which is occupied by the stomach and the colon and which yields frank tympany upon percussion when these viscera are not filled with solid material. Similarly, the inferior border of the spleen meets the external convex border of the left kidney at the level of the eleventh rib just external to the left scapular line, forming in this situation the *splenorenal angle,* which is occupied by the descending colon, yielding a tympanitic percussion sound upon percussion when this portion of the intestine does not contain solid material.

The presence of these tympanitic areas are of service in the delimitation of the spleen when they yield frank tympany; but not infrequently the stomach and intestine contain solid material and the angles yield a dull note which is suggestive of an augmentation of the volume of the spleen. Recognizing this possible source of error, Piorry always preceded percussion of the splenic region by thorough evacuation of the lower intestinal canal.

Owing to the intimate and continuous relation of the inferior border of the spleen with the superior pole of the left kidney internal to the left scapular line, it is impossible to establish by percussion the inferior limit of the spleen upon the posterior surface of the thorax. The superior limit of the organ is established by determining the inferior limit of pulmonary resonance in the scapular, postaxillary, and midaxillary lines; but here again, in the presence of effusion into the left pleural sac or in the presence of consolidation of the inferior lobe of the left lung it becomes impossible to establish the superior limit of splenic dullness and hypertrophy of the organ is apt to be suspected when it does not exist. A further source of error in splenic percussion is added when an enlargement of the liver causes the left extremity of the latter organ to invade the left hypochondriac region and to impinge against the spleen. In this event a false *splenohepatic angle* of tympany is established at the summit of Traube's semilunar space.

Percussion of the spleen has been practiced in all possible bodily attitudes in the hands of different clinicians. The subject has been placed in the abdominal position while the spleen was delimited upon the posterolateral aspect of the abdomen; he has been placed in the right lateral posture, which frequently causes the left iliac crest to impinge against the lower costal arch with obliteration of the left flank, which is a productive source of error; he has been placed in the right-lateral decubitus of Schuster; and Zeimssen has preferred to outline the spleen with the patient in the sitting posture. In the majority of cases the latter position is the preferable one during delimitation of the spleen by percussion. The right lateral decubitus may be employed if a pillow is slipped under the right side of the patient to prevent the left iliac crest from impinging against the left costal arch and thus disturbing the anatomic relations of the spleen.

In practicing auscultatory percussion of the spleen with the patient in the upright or sitting posture the examiner should apply the bell of the stethoscope in the tenth intercostal space in the scapular line; and, after fixing in his mind the quality and pitch of the percussion note in the neighborhood of the instrument, he should percuss upward toward the inferior border of the lung in lines drawn in arcs of a circle as described above, radiating toward the vertebral column, upward in the scapular line, and outward toward the posterior axillary line. Having fixed the inferior limits of pulmonary resonance in these lines, he should percuss downward in the same lines, whereupon he will find that the splenic dullness is continuous with the renal dullness to the iliac crest. External to the scapular line, however, upon percussing downward the examiner will find that at the inferior border of the eleventh rib the splenic dullness will give place to a tympanitic note in the posterior axillary, midaxillary, and anterior axillary lines, if the anterior extremity of the spleen reaches the last mentioned line. In delimiting the anterior extremity of the organ the examiner percusses from a region of frank splenic dullness near the bell of the stethoscope, in a direction radiating horizontally forward toward the median line of the abdomen. As a rule, when the costoarticular line is reached, the dullness of the spleen will give place to gastric tympany in Traube's semilunar space. It is very rare for the anterior extremity of the normal spleen to surpass this line. Leichtenstern has objected to the employment of the costoarticular line in forming conclusions as to hypertrophy and displacement of the spleen, for the reason that the course of this line depends entirely upon the conformation of the thorax, with which the spleen has no direct connection. This author points out that in a subject with an elongated thorax it is quite possible for the anterior extremity of the spleen to surpass this line in the absence of hypertrophy of the organ; whereas in the case of deep chested subjects the anterior extremity of the hypertrophied organ may fall short of this line.

Percussion of the splenic region should be practiced during tranquil respiration as deep inspiration is capable of modifying the superior limit of splenic dullness markedly and the superior diminution in the area is not attended by a corresponding increase in the inferior limits of the dullness. This discrepancy occurs through the fact that the respiratory mobility of the spleen is limited on account of the relatively small extent of the organ which is apposed to the inferior diaphragmatic surface as compared to that of the liver; and while the inferior border of the lung descends into the complementary sinus of the pleura to the extent of one to one and one-half inches during full inspiration, the spleen is not displaced downward to a corresponding degree. Indeed, Gerhardt established the facts that while during full inspiration the inferior border of the left lung descended to the extent of three to four centimeters, the inspiratory displacement of the inferior splenic border did not exceed one centimeter.

In the pathologic state the examiner may encounter an increase in the area of splenic dullness, a decrease in the extent of this area, or a total absence of splenic dullness upon percussion.

The moderate hypertrophies of the spleen which accompany the acute infectious diseases are scarcely to be detected during percussion of the organ. But in the presence of extensive chronic hypertrophy of the organ the area may embrace a large portion of the abdominal surface. In more moderate enlargements of the spleen there is a coincident extension of the superior and inferior limits of the organ with extension of the anterior extremity of the organ toward the median line of the body. Thus, it not infrequently happens that the anterior extremity of the spleen comes into contact with the left lobe of the liver, with the production of a continuous band of dullness in the hypochondriac and epigastric regions, with the formation of a *splenohepatic angle* of gastric tympany at the summit of Traube's semilunar space. Auscultatory percussion is serviceable in separating enlargements of the spleen from growths springing from other abdominal organs; and examination of the blood frequently reveals corroborative findings.

Attention has been called in a previous paragraph to the inherent difficulties of delimitation of enlargements of the spleen by percussion and the possibility of effusions into the left pleural sac and the presence of solid accumulations in the stomach and the colon simulating extensions in the various limits of splenic dullness.

Diminution of the area of splenic dullness is encountered with the maximum frequency in connection with hypertrophic emphysema, when the voluminous lungs encroach upon the superior limits of the area of splenic dullness. Similarly, in the presence of tympanites the spleen is crowded into the left vault of the diaphragm and the area of splenic dullness is greatly restricted or abolished. In the aged subject the spleen frequently undergoes a striking diminution in size, with consequent restriction of the area of splenic dullness.

Absence of splenic dullness is noted in the case of the wandering spleen, in which the spleen has fallen from its normal habitat, and in the rarer cases of congenital absence of the organ. In the presence of pneumoperitoneum the collection of gas may insinuate itself between the spleen and the abdominal wall and temporarily abolish the dullness of the spleen.

Before basing conclusions as to the probable dimensions of the spleen upon the percussion findings, the student should pass in review the various factors which may influence these findings, and which have been enumerated in the foregoing paragraphs; and a diagnosis of splenic hypertrophy should not be formulated in any case in which the spleen does not become palpable.

THE KIDNEYS

In the physical examination of the kidneys, percussion is inferior to palpation as a method of examination, and its employment with satisfactory results is practically confined to the study of enlargements of the organs. In the case of the normal kidney it is impossible to map out any area of renal dullness upon the anterior surface of the abdomen, and it is exceedingly difficult in the case of the normal organs to establish any area of renal dullness upon the posterior abdominal wall which bears any definite anatomic relation to the true position of the kidneys. In the case of the normal kidneys, the superior limits of the organs are always obscured by their relations with the liver and with the spleen. It is equally impossible to delimit by percussion the internal concave borders of the organs on account of the thick musculature occupying the areas upon either side of the vertebral column and because of the fact that the transverse processes of the vertebræ frequently intervene between the internal borders of the organs and the posterior abdominal wall.

Externally it is possible, when the colon does not contain solid fecal accumulations to establish along the external border of the thick bed of muscles paralleling the vertebral column a limit where the so-called renal dullness gives place to intestinal tympany. This limit is commonly accepted as the external limit of renal dullness, and is assumed to represent the external border of the normal kidney; but Weil established the fact that this limit corresponds merely to the free edge of the massive muscular column erected upon either side of the vertebral column and in no wise represents the anatomic position of the external border of the normal kidney. For the same anatomic reason the examiner will very rarely succeed in delimiting the inferior pole of the kidney, as it is covered by the thick superjacent musculature, which effectually deadens any tympanitic sound which might be generated in the intestine at the level of the inferior pole of the kidney. Thus, percussion of the kidney is practically limited to the delimitation of renal enlargements, in which the hypertrophied organ approaches the anterior abdominal wall, thereby enabling the examiner to base his conclusions upon certain regional variations in the percussion sounds elicited upon this abdominal surface.

Here renal growths are differentiated from hepatic hypertrophies by the absence of respiratory mobility, by the fact of their carrying the tympanitic colon in advance of them, and by the impossibility in the case of hepatic growths of insinuating the finger tips between the growth and the ventral abdominal wall.

Similarly, growths springing from the spleen exhibit respiratory mobility; they are situated higher up in the left hypochondriac region than are renal growths; while the finding of the colon in front of the growth pleads in favor of a renal growth, as growths of the spleen develop above the level of the colon, which they displace downward.

Renal growths of extensive dimensions may be mistaken for growths of the ovary. In this connection the examiner should recall that renal growths develop from above downward, while tumors of the ovary develop from below upward; that renal tumors are ordinarily encountered behind the intestines and carry the tympanitic colon forward before them, while ovarian tumors are situated in front of the intestine; and that renal tumors are attended by urinary changes, while ovarian growths provoke menstrual disturbances and uterine displacements.

THE BLADDER

Percussion is available only in cases of extreme distention of the bladder, when a flat note is elicited over the distended bladder in the superpubic and umbilical regions surrounded by a zone of intestinal tympany.

THE STOMACH

Percussion of the stomach is employed to determine the size, shape, and position of the viscus. During the examination, as in the case of palpation and inspection of the epigastrium, the subject should occupy the dorsal decubitus. In the examination of the organ by percussion, finger percussion, flicking percussion or auscultating percussion may be employed with uniformly good results in the course of the examination.

The findings upon percussion of the stomach vary with the state of the organ. The sound which is elicited depends upon the contents of the stomach and upon the tension of its walls. According to the different variations in these factors the gastric cavity is capable of generating pure tympany, a metallic sound, or frank flatness. As the stomach is continually presenting active contractions and relaxations, the percussion sound which is elicited naturally changes with great frequency during a brief period of time.

Pure gastric tympany is only obtained in Traube's semilunar space, when the stomach is moderately distended and its walls are not under undue tension. This area, corresponding to the portion of the anterior gastric wall which is in direct apposition with the thoracic and abdominal surface, presents a transverse diameter of four to five inches and a vertical diameter of three to four inches. The space possesses three distinct limits which may be determined by alterations in the quality and pitch of the percussion sound. Superiorly and to the left the gastric tympany is interrupted by the inferior border of the left lung, the *gastropulmonary limit* of the space; superiorly and toward the right it is interrupted by the left lobe of the liver, the *gastrohepatic limit;* while inferiorly it is interrupted by the colonic tympany of the transverse colon, constituting in this situation the *gastrocolic limit* of gastric tympany. In the minimal number of cases in which the tip of the left lobe of the liver does not reach the apex of the heart, there exists yet another change in the percussion sound in this situation, constituting a *gastrocardiac limit* of Traube's space.

The gastropulmonary and gastrohepatic boundaries of the semilunar space of Traube are readily determined by mediate percussion with the fingers from an area of frank gastric tympany in the central portion of this area toward the lung and the liver respectively. The delineation of the gastrocolic boundary of the space is more difficult, owing to the fact that it is possible for the gastric and the colonic tympany to approximate one another in quality and pitch. This difficulty is obviated by the introduction of a quart of fluid into the stomach prior to the examination. Upon percussing upward under these conditions, the colonic tympany gives place to dullness or flatness, when the greater curvature of the stomach is reached.

Auscultatory percussion is to be preferred, however, in the accurate determination of the size, shape, and position of the stomach. In this method of examination the chestpiece of the stethoscope is applied at the center of gastric tympany in Traube's space. The examiner delivers a few blows and fixes in his mind the quality and pitch of the sound elicited. Without moving the position of the bell of the instrument, he then begins at several points upon the abdominal surface and percusses toward the bell of the stethoscope. In each instance a change in the quality of the percussion note will indicate when the gastric border has been reached. Auscultating percussion along arcs of a circle as noted elsewhere is particularly valuable in outlining gastric boundaries.

Scratching percussion is also very serviceable in delimiting the borders of the stomach. In this procedure the examiner applies the bell of the stethoscope to the area of frank gastric tympany in Traube's semilunar space and scratches the skin of the abdomen in this area with the nail. Having fixed in his mind the quality and pitch of the sound elicited, he begins at various regions of the abdominal surface and passes the finger-nail along the skin toward the bell of the instrument. A change in the quality of the sound is noted in each instance when the gastric borders are reached. The rubber tip of the percussion hammer is very serviceable in scratching percussion, as is also the eraser upon the end of a lead pencil, which is produced upon percussion in Traube's semilunar space. In each instance the quality of the sound changes abruptly when the gastric borders are attained.

An *extension* of the area of gastric tympany does not in every case in which it is encountered indicate an increase in the volume of the organ. In the presence of atrophic changes in the liver the left lobe of this organ is retracted toward the median line and the gastric tympany is extended in this direction, with the consequent production of a gastrocardiac limit of the space of Traube. Similarly, in the presence of retraction of the anterior and inferior borders of the left lung in the course of chronic interstitial pneumonia, fibroid phthisis, or pulmonary syphilis, the gastric tympany is extended in a direction upward and outward as a result of retraction of the gastropulmonary limit of this space.

Extension of the area of gastric tympany as a result of enlargement of the organ itself is encountered in the presence of gastrectasis and in congenital enlargement of the organ, (megalogastria). In gastroptosis, on the contrary, although the inferior limit of the area of gastric tympany is markedly lowered, it can be determined by careful percussion that the lesser curvature no longer resides in its normal habitat beneath the left lobe of the liver. Again, in the case of gastrectasis, an error is apt to arise through the accumulation of fluid in the dilated stomach if care is not exercised to evacuate this fluid prior to the examination.

Diminution of the extent of gastric tympany is by no means invariably associated with diminution in the size of the stomach. In hepatic enlargements the left lobe of the liver encroaches upon the semilunar space of Traube and causes downward displacement of the gastrohepatic limit of this space. Similarly, in the presence of left-sided pleural effusions and pyopneumothorax, the gastropulmonary limit of the space is lowered with consequent diminution in the limits of gastric tympany. Pitres has noted diminution in the dimensions of gastric tympany in connection with extensive effusion into the right pleural sac, which he attributes to crowding over of the liver by the weight of the effusion. The presence of varying quantities of solids and fluids in the stomach diminishes the area of gastric tympany.

Diminution of the area of gastric tympany which is referable directly to diminution in the size of the stomach is occasionally demonstrable in the presence of cardiac stenosis from malignant disease of the cardiac orifice of the stomach. However, in this event the atrophic stomach as a rule is pushed upward and backward by the transverse colon and in no wise enters into the tympany which is produced upon percussion in Traube's semilunar space. Indeed, Dehio has demonstrated, both upon the living subject and upon the cadaver, that when the normal stomach is empty the tympany which is obtained upon percussion of Traube's space is invariably due to the transverse colon, which crowds the empty stomach upward into the left concavity of the diaphragm.

An hourglass constriction of the stomach may be demonstrated by the introduction of water into the stomach; when, upon percussion over the viscus, it is observed that the stomach is not uniformly distended; but that the cardiac portion is distended, while the pyloric portion remains empty; and, moreover, that in a short time fluid passes into the pyloric portion, which in turn becomes distended. If, during this time, the stethoscope be applied over the central portion of the stomach, it may be possible to hear the water gurgle through the constricted portion of the organ.

THE SMALL INTESTINE

The note which is elicited upon percussion of the small intestine varies in pitch and quality with the amount of gas contained in the tube and with the tension of its walls. The normal intestine yields a tympanitic or metallic percussion sound, but in the presence of a considerable content of solid material, the note becomes dull. When the intestine is greatly distended with gas in intestinal obstruction, the note is frankly tympanitic, but of slightly lower pitch than in the case of percussion of the normal intestine with only moderate tension of its walls. A similar percussion note is encountered in the presence of a solid tumor which is surrounded by tympanitic intestine. The same note is encountered in the presence of multiple incomplete stenoses of the intestine as the result of tuberculous ulceration. Frequently under these circumstances there is accumulation of fluid above the level of each of the obstructions, yielding impairment of intestinal tympany in multiple areas of the abdominal surface, constituting the sloping dullness of Mathieu and Ricard.

THE COLON

As in the case of the small intestine, the findings upon percussion of the colon are variable and extremely difficult of correct and accurate interpretation. The note elicited upon percussion of the colon containing gas is frankly tympanitic or metallic, its quality and pitch varying with the tension of the colonic walls. Yet it is practically impossible at the bedside to differentiate the percussion sounds elicited upon percussion of the stomach, the small intestine, and the colon with any degree of accuracy, as the quality and pitch of the sounds vary with variations in the tension of the walls of the various portions of the intestinal tract. It is possible for coils of the small intestine, in spite of their lesser size and capacity, to yield a tympanitic note quite analogous to that which is generated in the moderately distended colon. The tympany of the transverse colon is differentiated from that of the stomach with most ease when the latter organ is partially filled with fluid. The presence of fecal accumulations in the various portions of the large intestine masks the tympanitic quality of the percussion note, yielding varying degrees of dullness upon percussion.

SECTION VI

AUSCULTATION

CHAPTER XXVI

OBJECT AND TECHNIC OF AUSCULTATION

Auscultation was practiced by Hippocrates in the study of certain affections of the thoracic viscera, notably in the diagnosis of hydropneumothorax by the elicitation of the succussion sound which bears his name. He was also familiar with the pleural friction sound in plastic pleurisy, and described certain types of râles or "rattles" in the bronchial tubes. In this day it seems almost incomprehensible that these fundamental principles of physical diagnosis should have been neglected for centuries. Again, in the latter part of the seventeenth century, Robert Hooke, an English physician and a contemporary of Newton, practiced auscultation of the thorax; but his work likewise failed to awaken a lasting interest in the method.

In the early part of the nineteenth century we find Corvisart in France practicing immediate auscultation of the sounds of the heart, but he apparently did not extend his studies to diseases of the bronchopulmonary system.

Mediate auscultation was first practiced in Paris by Laënnec, whose work entitled "Traité de l'auscultation médiate," in which he set forth his experience with the method during three years, appeared in 1819. The stethoscope of Laënnec was a monaural instrument made of wood, and was equipped with a chest-piece not unlike that of the modern bell type of binaural stethoscope.

Following Laënnec, Skoda of Vienna, who had attained distinction as one of the early elaborators of the art of percussion, contributed valuable data to the new art of auscultation, devoting particular attention to auscultation of the heart sounds and endocardial murmurs.

Auscultation, the act of listening to sounds which are generated within the body, may be practiced with the unaided ear, or with a specially devised instrument, the stethoscope. When the ear is employed, the procedure is termed *immediate auscultation* in contradistinction to instrumental auscultation, which is termed *mediate auscultation.* The method is employed in the study of the lungs, heart, and abdominal organs, and in eliciting certain vascular phenomena.

Stethoscopes are monaural and binaural, as they are equipped with one or two ear-pieces, respectively. The monaural instrument is seldom employed by American clinicians, having yielded place to several types of binaural stethoscope. The modern binaural stethoscope consists of a bell or chest-piece of hard rubber or metal, which is connected with the ear-pieces by rubber tubing, the principle involved being the clear transmission of sound from the subject to the ear of the examiner and the exclusion, as far as possible, of extraneous noises.

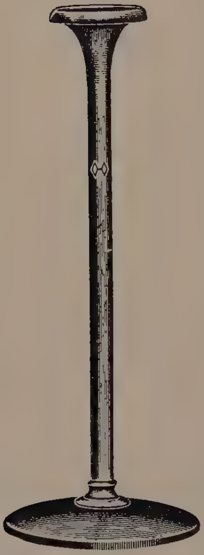


Fig. 209.—Hawksley's monaural stethoscope.

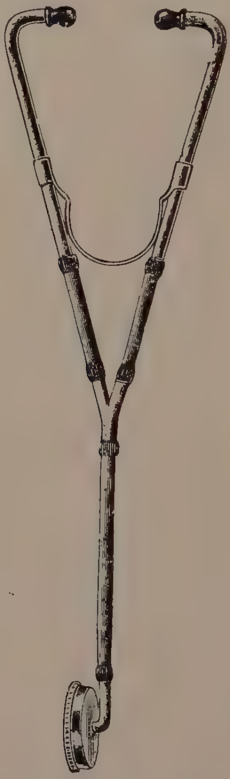


Fig. 210.—Bowles stethoscope.

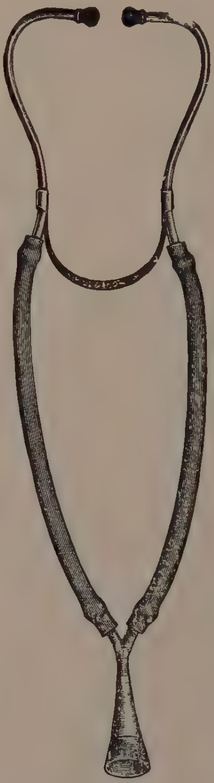


Fig. 211.—Binaural stethoscope.

In the Bowles stethoscope the bell is replaced by a chest-piece containing a vibrating diaphragm. This type of instrument is very useful in examining bed-ridden patients, and when for any reason it is necessary to slip the chest-piece beneath the clothing. The instrument is also very serviceable in auscultation of the peripheral arteries in blood-pressure estimations.

Various types of stethophones have been employed in the practice of auscultation, the object of the instruments being to magnify the intensity of the sounds which are elicited; but their use should be guarded, as they are poor conductors of high-pitched sounds, and from a clinical standpoint the clearness and quality of the sounds are of greater importance than is an artificial magnification of the intensity of the sounds elicited.

In the selection of a stethoscope, whatever type is adopted, care should be exercised to secure an instrument whose bell and tubing are sufficiently heavy to exclude extraneous noises, and whose ear-pieces fit snugly in the ears of the examiner. Again, heavy tubing tends to prevent kinking of the tubes during the examination.

Immediate Auscultation.—In the practice of immediate auscultation, the ear of the examiner is applied directly to the part under examination, only a thin garment or a towel intervening between the ear and the chest wall. The position of the patient should conform to that which is described during mediate auscultation. Aside from objections from the esthetic point of view, immediate auscultation of the thorax yields sounds from so extensive an area that exact localization of the signs is difficult in many cases.

Mediate Auscultation.—In the practice of mediate auscultation the bell of the stethoscope is applied firmly and evenly to the chest wall, but without the exertion of undue pressure. The bell of the instrument is retained in position by grasping it near the base with the index finger and the thumb. No article of wearing apparel should be permitted to intervene between the chest-piece of the instrument and the surface of the thorax.

Before practicing auscultation of the thorax the nares should be examined with the view of detecting obstructions to the passage of air during respiration. If such an obstacle to normal respiration exists, it is desirable to practice auscultation while the patient breathes through the open mouth. Similarly, in persons who habitually breathe after the costoabdominal method, it is sometimes necessary to have them practice costal respiration to bring out the respiratory sounds over the upper portion of the thorax.

Auscultation is practiced by preference with the patient in the sitting posture. During auscultation of the anterior surface of the thorax the arms of the patient should hang naturally at the sides, and his position should be natural and free from undue muscular tension. In auscultation of the axillary and infra-axillary regions the patient should raise the arms laterally only so far as is necessary to insure a free access to this portion of the thorax. During auscultation of the posterior aspect of the thorax the patient should incline the trunk forward and clasp the arms across the chest to insure a wide separation of the scapulæ.

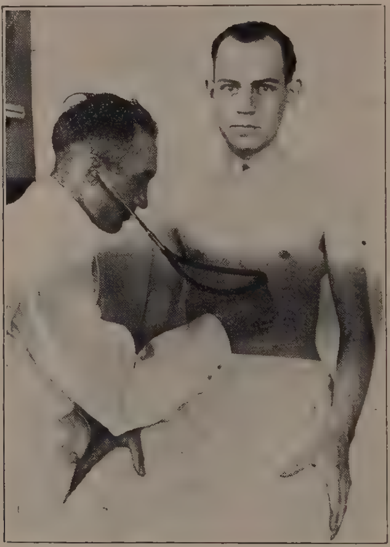


Fig. 212.—Auscultation of thorax.

Auscultation of the thorax should be practiced methodically, the examiner progressing downward from the apices to the bases of the lungs. In each instance the sound which is elicited upon one side should be compared with the sounds elicited over the corresponding area upon the opposite side of the thorax. Wandering and hasty auscultation of the thorax, without comparison of the sounds upon the two sides, is a productive source of error in physical examinations.

The ear of the examiner should be trained to disregard all extraneous noises, such as the friction produced by the rubbing together of the tubes of the instrument, the contact of the hand with the instrument, and crepitation due to contact of the bell of the stethoscope with a hairy chest wall. This last annoying feature may be eliminated by moistening the bell of the instrument prior to its application to a hairy chest wall. Tipping of the bell or chest piece sufficient to allow a layer of air to come between the chest wall and the chest piece of the stethoscope interferes with sound transmission, and also at times changes the character of the sounds. The beginner shoud listen for some time over a given point before changing the location of the stethoscope. It is surprising how much more clearly sounds are heard after even a few seconds.

After becoming familiar with sounds produced within the stethoscope and by faulty contact or rubbing on the skin, the beginner should try to familiarize himself with sounds produced by contraction of skeletal muscles. This is best accomplished by placing the chest piece of the stethoscope over a thick band of muscle, such as the trapezius or biceps, and listening while the muscle is at rest and when it is forcibly contracted. The contracting muscle produces a peculiar roaring sound, often accompanied by crackles, sometimes difficult to distinguish from râles.

The beginner in auscultating usually experiences some difficulty in separating the pulmonary and the cardiac sounds; but concentration and practice will enable him to disregard the one while studying the other.

During auscultation of the respiratory organs the examiner should note the character of the sounds which are generated during quiet and during forced respiration, and again during inspiration following cough. During quiet respiration in the normal subject little or no expiration is audible, whereas during forced respiration there is an undue prolongation of this phase of the respiratory cycle. Râles which are inaudible during quiet breathing are frequently brought out by deep inspiration, especially by deep inspiration following expiration and a short cough.

CHAPTER XXVII

AUSCULTATION OF THE RESPIRATORY SYSTEM

NORMAL RESPIRATORY SOUNDS

Upon auscultation of the various regions of the thorax of the normal subject three types of respiratory murmur are encountered; namely, *bronchial breathing, vesicular breathing,* and *broncho-vesicular breathing.*

Bronchial Breathing.—Bronchial breathing is a blowing, tubular sound, the expiratory phase of which is usually longer than inspiratory, although occasionally they are of equal duration. The inspiratory and expiratory phases of the murmur are separated by a distinct interval; and the intensity and pitch of the two phases are different, for physical reasons which will be detailed in succeeding paragraphs.

Bronchial breathing in its maximum purity and intensity is elicited upon auscultation of the larynx and trachea, over the site of production of the aerial whorls which call the sounds into being. The sound is also commonly audible in the interscapular regions in the area situated between the seventh cervical and fourth dorsal vertebra, and over the manubrium sterni anteriorly, the latter two levels corresponding to the level of the tracheal bifurcation. These are the regions of the thorax in which bronchial breath sounds are encountered during tranquil respiration. In the presence of extreme grades of dyspnea, with marked increase in the force and depth of the respirations, it is not uncommon for bronchial breath sounds to become audible over the entire thoracic surface.

When during inspiration the inspired air passes through the orifice of the glottic slit and enters the wider cavity of the larynx beyond, aerial whorls are generated in the air content of this cavity, whorls which are conducted downward into the trachea and larger bronchi in the form of the inspiratory phase of bronchial breathing. Similarly, when during expiration the expired air passes through the chink of the glottis into the broader pharyngeal cavity beyond, similar whorls are induced with the production of the expiratory phase of bronchial breathing.

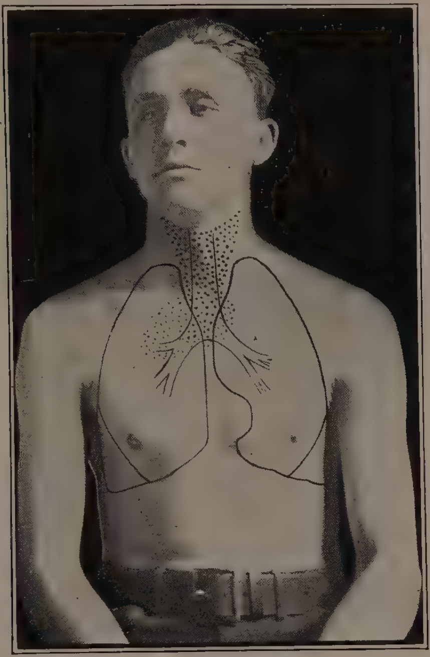


Fig. 213.—Normal distribution of bronchial and bronchovesicular breathing. Anterior thoracic surface.

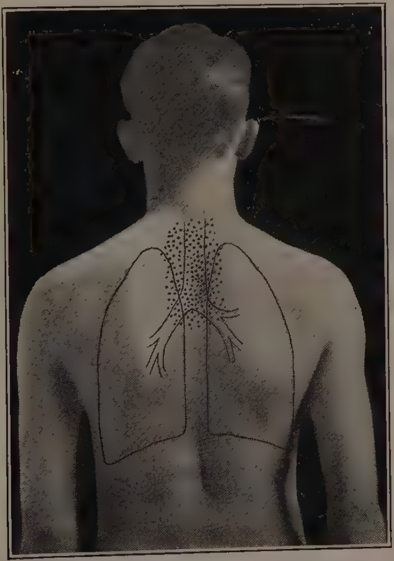


Fig. 214.—Normal distribution of bronchial and bronchovesicular breathing. Posterior thoracic surface.

As the genesis of the sound in each instance lies in the passage of air through the glottic slit, it is easy to comprehend the influence of the diameter of this opening upon the intensity and the pitch of the two phases of bronchial respiration. The physical laws governing aerial currents teach us that the intensity of the sound engendered by such currents varies in direct proportion with the degree of stenosis or narrowing of the orifice. Hence, the expiratory phase of bronchial respiration, corresponding to the narrowing of the glottis at this time, is more intense than the inspiratory phase of bronchial breathing. Similarly, since according to the physical laws governing the pitch of sounds generated in this manner the pitch is in direct proportion to the size of the opening, one readily understands that the inspiratory phase of bronchial breathing, corresponding to the widening of the chink of the glottis during inspiration, is of distinctly lower pitch than is the expiratory phase.

The tendency for these vibrations to be conducted downward throughout the bronchial system and outward to the thoracic wall by way principally of the aerial content of these tubes and secondarily by the walls of the tubes themselves, is combated by the great mass of aerated pulmonary parenchyma which intervenes between the principal bronchi and the thoracic walls. In the presence of extensive consolidation of the pulmonary alveoli, however, and in the presence of extensive cavitation of the lung with free communication with the bronchial system, the bronchial sounds generated in the larynx are conducted to the thoracic wall with an intensity and clearness which justifies the descriptive term of "tubular breathing."

Vesicular Breathing.—Upon auscultation of the infraclavicular and mammary regions anteriorly, of the axillary and infraaxillary regions laterally, and of the infrascapular regions posteriorly, regions of the thoracic surface which overlie portions of the lungs which are comparatively remote from the principal bronchial tubes, the respiratory sounds possess a soft, breezy sound of low pitch, which is termed vesicular or alveolar breathing. The murmur consists of two phases, inspiratory and expiratory, which are separated by a short interval. The inspiratory murmur is maintained two or three times as long as is the expiratory murmur. The quality of vesicular breathing has been compared to the sound produced by the soft rustling of the leaves of a tree when agitated by a gentle breeze. The quality of the sound can also be rather closely simulated by approaching the lips nearly in contact and inspiring strongly.

The physical genesis of the vesicular murmur has not as yet been accounted for in an adequate manner. Laënnec erroneously attributed the sound to the rubbing of the inspired and expired air against the walls of the bronchi and pulmonary alveoli, an explanation which rests upon no adequate physical basis. Blakiston held that during inspiration the involuntary muscular fibers, disposed in a circular manner around the walls of the smaller bronchioles, contracted with the consequent production of partial stenosis of these tubules with the generation of the vesicular murmur, a theory which is not substantiated entirely from either the physiologic or physical point of view. Leaning would have us believe that the murmur is a purely muscular sound, generated by contraction and relaxation of the smooth muscle bands of the bronchioles during inspiration and expiration. Gerhardt attributed the vesicular murmur to vibrations of the alveolar walls during inspiration. At the present time the most probable explanation of the sound, it would seem, is that of Baas and Penzoldt, who state that the vesicular murmur is merely bronchial breathing softened in its transmission from the larynx along the aerial column contained in the trachea and bronchi, mingled with the soft crepitation of innumerable pulmonary alveoli during their inflation and subsequent collapse.

The prolongation of the inspiratory portion of the vesicular sound is more apparent than real, being actually somewhat shorter than the inspiratory murmur of bronchial breathing. The fact that the vibrations are carried down the trachea with the inspired air lessens reflection and helps to preserve both length and volume. The expiratory murmur on the contrary must travel against the current of expired air, increasing reflection, and thus materially diminishing both length and volume.

Whatever may be the precise genesis of the vesicular breath sound, clinical experience has taught us to construe its presence to mean that in any region of the thorax where it is encountered, the pulmonary alveoli and bronchioles are permeable to air. It does not follow from this, however, that its presence assures a normal condition of these structures. In the small, disseminated patches of consolidation attending miliary tuberculosis of the lungs and lobular pneumonia it is possible for the intervening aerated areas of the lung to suppress any enfeeblement of the murmur which might arise as the result of the exclusion of the air from the minute and scattered areas of alveolar consolidation.

The intensity of the vesicular murmur is dependent upon the force of the respiratory efforts and the thickness of the thoracic walls in the normal subject. The influence of the depth of the respiratory act upon the murmur is well illustrated upon auscultation of the thorax during Cheyne-Stokes respiration, when the intensity of the vesicular murmur will exhibit all gradations from accentuation of the murmur at the height of the dyspneic period to its total abolition during the period of comparative apnea. In children and in the female subject, owing to the thinness and elasticity of the thoracic parietes the vesicular murmur is more intense than is the case in the adult male subject. The age has a direct bearing upon the intensity of vesicular breathing. Up to the twelfth year of life, vesicular respiration is more intense than it is subsequent to that age; while in the aged subject there is a progressive diminution in the vesicular element of the respiratory sounds, which come to approach the quality of bronchial breathing or bronchovesicular breathing.

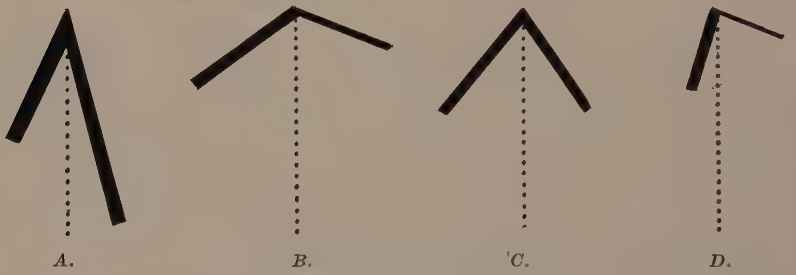


Fig. 215.—*A*, Very loud bronchial breathing; *B*, exaggerated vesicular breathing; *C* and *D*, two common types of bronchovesicular breathing. (Redrawn from Calbot.)

The line to the left of the dotted line represents inspiration, that to the right expiration. The width of the line represents intensity, the length represents the length, and the angle with the dotted line the pitch.

The pitch of vesicular breathing varies with the age and sex of the subject. It is uniformly of higher pitch in the infant and young child than it is in the adult subject; and it is of higher pitch in the female than in the male subject. The variation in the pitch of the murmur is in each of these instances caused by the smaller diameter of the glottic slit in the first class of subjects. Again, in the aged subject, without regard to sex, there is an increase in the pitch of vesicular breathing, the result in this instance of senile rarefaction of the pulmonary tissues.

Bronchovesicular Breathing.—This type of respiration, as its name implies, combines the qualities of bronchial and of vesicular breathing. The inspiratory phase closely resembles the normal vesicular murmur, while the expiratory phase, the duration of which may equal the inspiratory phase, has a tubular quality engrafted upon it. It is audible over regions of the normal thorax in which the larger bronchi are in fairly close proximity to the thoracic wall, but are nevertheless covered by aerated pulmonary tissue. Thus, bronchovesicular breathing is normally audible over the lower portion of the manubrium sterni and adjacent to its right lateral border anteriorly, and in the interscapular regions posteriorly at the level of the fourth dorsal vertebra.

Calbot suggests convenient diagrams to illustrate the different types of breath sounds.

ABNORMAL RESPIRATORY SOUNDS

In the presence of disease of the respiratory organs, various modifications of the normal respiratory sounds are encountered. The alteration may consist of the presence of bronchial or bronchovesicular breathing in a region of the thorax where it is not normally present; of the assumption by bronchial breath sounds of certain cavernous or amphoric qualities which are not normally attributable to them; of variations in the intensity of the vesicular murmur in regions of the thorax where it is normally present; or of variations in the frequency or rhythm of the respiratory sounds.

Bronchial Breathing.—When bronchial breath sounds are detected in a region of the thorax where they are not normally audible, it most frequently points to consolidation of the lung, the solidification conducting the murmur from the large bronchi to the surface of the thorax. Other factors which act similarly comprise pulmonary compression and collapse, hemorrhagic infarction, cirrhosis of the lung, enlarged bronchial glands, a tumor overlying a large bronchus, or a pulmonary cavity situated near the chest wall with a free bronchial communication.

Bronchial breathing which is referable to a cavity with patent bronchial communication frequently has a peculiar hollow quality engrafted upon it to which the term *cavernous breathing* is applied. In this subtype of bronchial breathing expiration is frequently of lower pitch than is inspiration. Similarly, a cavity with patent bronchial communication or a pneumothorax with an open bronchial fistula often gives rise to bronchial breathing of rather musical quality, closely simulating the sound which is generated by gently blowing across the mouth of an empty bottle, *amphoric breathing.*

Over the site of a tuberculous consolidation or pulmonary cavity there is occasionally to be elicited a type of breathing which begins as a vesicular, and terminates as a bronchial sound.

A further subtype is characterized by an inspiratory murmur which begins as bronchial breathing, but which terminates as cavernous or amphoric breathing. This form of inspiratory murmur is occasionally to be elicited over the site of a pulmonary cavity with a small patent bronchial communication. Forced inspiration must be practiced in the elicitation of the sound.

Vesicular Breathing.—In disease of the respiratory organs the intensity and rhythm of the normal vesicular murmur are frequently so altered as to possess definite diagnostic significance.

Diminution in the intensity or entire abolition of the vesicular murmur is normal in aged subjects and in subjects with very thick chest walls. The murmur is also diminished in the presence of painful diseases of the chest wall such as incipient pleurisy and pleurodynia, which cause the patient to inhibit the respiratory excursions of the chest. A similar diminution occurs with moderate pleural thickening, edema of the lung, the early stage of lobar pneumonia, and in the presence of a closed pneumothorax.

Abolition of the vesicular murmur is noted over a region of the thorax in which the main bronchus is occluded, over a large pleural effusion, and over a pulmonary cavity which is filled with fluid.

Increased Intensity (Puerile Breathing).—Exaggeration of the vesicular murmur is noted over a lung which is the site of compensatory emphysema due to crippling of the opposite lung, over a circumscribed portion of a lung which is expanding vicariously to compensate for consolidation in an adjacent focus, in catarrhal inflammations of the smaller bronchioles, and during the dyspnea of imperfectly compensated cardiac disease.

Prolongation of the expiratory phase of the vesicular murmur accompanies hypertrophic emphysema and bronchial asthma. In these states the alteration in the phases of the sound is noted over both sides of the thorax; and in the case of asthma expiration is dotted with râles. Unilateral prolongation of the expiratory phase at an apex is suggestive of incipient pulmonary tuberculosis, particularly if noted at the left supraclavicular fossa.

Cog-wheel Breathing.—In certain diseases of the respiratory organs the respiratory murmur, and particularly the inspiratory phase of the murmur, occurs in a series of short gasps or jerks, closely simulating the sound emitted by a sobbing child. This jerking or cog-wheel modification of the vesicular murmur is a valuable sign of incipient phthisis. It is noted with far less constancy in hysteria, bronchial asthma, chorea, local catarrhal conditions of the bronchioles, in the pain of a fractured rib, pleurodynia, and pleurisy.

Bronchovesicular Breathing.—When encountered in an area of the thorax where it is not normally audible, this type of respiration points to a moderate degree of the same pathologic changes which produce frank bronchial breathing. It is a sign of partial or incomplete consolidation, as in the early stage of pneumonia or phthisis, or of a cavity or a solid tumor of the lung which is covered by normal crepitant pulmonary tissue.

VOCAL RESONANCE

Vocal resonance is the transmission of inarticulate or articulate sound to the ear of the examiner upon auscultation of the thorax during the act of phonation. In eliciting the phenomena of vocal resonance, the bell of the stethoscope is applied firmly and evenly to the surface of the thorax while the patient is directed to count "One, two, three," or to repeat the words "ninety-nine" in a voice of uniform intensity and with the face turned from the examiner. According to the aim of the examination, the patient repeats the words either aloud or in a whisper. In either event, it is essential that the intensity of the voice remain uniform throughout the examination. In the course of the examination the intensity of the sounds elicited from various regions of the thorax should be studied, and the intensity upon symmetrical regions should be carefully compared.

The intensity of vocal resonance presents regional variations in the various regions of the thorax which correspond accurately to the regional variations in the intensity of vocal fremitus in the normal subject. As in the case of vocal fremitus, vocal resonance is elicited in its maximum intensity and purity upon auscultation of the larynx. Upon auscultation over the thyroid cartilage the sound is so intense that it is frequently painful to the ear, *laryngophony.* Moreover, the quality of the voice as appreciated upon auscultation is observed to have quite completely altered in quality. It possesses upon auscultation a distinct nasal quality, which is rather closely simulated when the subject speaks with the nares closed or with a membrane stretched tightly across the lips.

The causes of the alteration in the quality of the voice are multiple, being due to the propagation of the vocal vibrations across the solid cartilages of the larynx instead of through free air; to the fact that the propagation does not follow the direction of the molecular oscillations, but occurs perpendicularly to them; and to a simultaneous mingling of the vibrations of the laryngeal cartilages with the vocal vibrations (Eichhorst).

Upon auscultation of the median line of the root of the neck and the episternal notch, corresponding to the course of the trachea, the vocal vibrations possess great intensity, *tracheophony,* but which does not equal the purity of the resonance as elicited over the larynx. Again, upon auscultation of the interscapular region at the level of the fourth dorsal vertebra, overlying the origin of the principal bronchi, vocal resonance is also extremely pure, *bronchophony.*

Upon auscultation of the infraclavicular, mammary, axillary, infraaxillary, and infrascapular regions, regions of the thorax which overlie crepitant pulmonary tissue in which the larger bronchi are comparatively remote from the thoracic surface, the examiner appreciates in the normal subject only certain rumbling, inarticulate sounds, which arise in the vocal cords and are transmitted downward into the lung and to the thoracic surface by the air column of the trachea and bronchi and the pulmonary parenchyma.

As in the case of vocal fremitus, again, the intensity of vocal resonance depends upon the intensity and pitch of the voice, and upon the thickness and elasticity of the thorax; whence it follows that it is relatively less intense in the case of young children and in the female subject. In the aged subject, on the contrary, owing to wasting of the investing musculature of the thorax and to the progressive calcification of the bronchial cartilages, vocal resonance becomes progressively more intense with increasing age. In this class of subjects the sound very frequently assumes a quavering, nasal quality, analogous to egophony in younger subjects, on account of the natural quaver of the senile voice.

Pathologic Variations

As the intensity of vocal fremitus is modified by various lesions of the thoracic viscera, so also vocal resonance, the auscultatory equivalent of vocal fremitus, under similar circumstances exhibits variations which, in the main, are very similar and closely akin to the variations in vocal fremitus. In addition, in the presence of certain diseases of the bronchopulmonary system, vocal resonance exhibits definite and more or less pathognomonic modifications in its quality.

Diminution or Absence.—The intensity of vocal resonance is impaired in the presence of hypertrophic emphysema or compensatory emphysema, owing to the distention of the lung incident to these conditions. It is similarly diminished or abolished in the presence of pleural thickening, moderate pleural effusion, and bronchial stenosis. A pulmonary cavity containing fluid causes abolition of vocal resonance in the area in which it lies. While in the case of pleural effusion the usual finding is abolition of vocal resonance over the distribution of the fluid and exaggeration of the resonance above the limit of the fluid, yet in the presence of pleural adhesions which traverse the fluid effusion, it is possible for vocal resonance to be manifest in circumscribed portions of the thoracic surface overlying the effusion.

Increased Vocal Resonance.—Vocal resonance is increased by the same factors which cause an increase of vocal fremitus; namely, consolidations, pulmonary compression, and cavities with free bronchial communication. The various gradations of increased vocal resonance are designated by different names.

Bronchophony is a form of increased vocal resonance in which the transmitted voice is very distinctly audible, sounding as if it were very near the ear. However, the speech is not articulate as it is in the next ascending grade, pectoriloquy. Bronchophony points to dense consolidation, particularly to consolidation which overlies or is superimposed upon one of the principal bronchi.

Pectoriloquy, the transmission of the articulate voice upon auscultation, is evidence of a very dense consolidation overlying a principal bronchus, or of a pulmonary cavity or pneumothorax with free bronchial communication. With less frequency pectoriloquy is elicited upon auscultation of a portion of the lung which is compressed by a pleural effusion. In the cases in which the voice is transmitted with great distinctness it is very suggestive of a pulmonary cavity.

Whispering pectoriloquy, the transmission of the articulate whisper upon auscultation, represents the highest refinement of vocal resonance. The articulate whisper is commonly audible in the normal subject immediately over the trachea and the larger bronchi in the inner portion of the right infraclavicular region anteriorly and in the upper interscapular regions posteriorly. When it is elicited elsewhere it is a very good sign of a dense pulmonary consolidation which is traversed by a bronchus, of a superficial pulmonary cavity with free bronchial communication, or of a pneumothorax with communicating bronchial fistula.

In the presence of the apical consolidation of chronic phthisis whispering pectoriloquy frequently assumes a quavering, muffled quality, which is very suggestive of the underlying pathologic condition.

D'Espine's Sign.—D'Espine demonstrated that in the presence of enlargement of the tracheobronchial glands, auscultation of the whispered voice over the spines of the cervical and dorsal vertebræ yields an articulate whisper below the level of the seventh cervical spine, whereas in the normal subject articulate sound is not appreciated below this level.

MODIFIED VOCAL RESONANCE

Egophony.—In the presence of moderate pleural effusion, upon auscultation of the thorax immediately above the level of the fluid during phonation, the vocal sounds are apt to assume a peculiar, quavering, nasal tone, somewhat resembling the plaintive bleat of a goat. This peculiar sound constitutes the egophony of Laënnec. Most readily elicited posteriorly near the angle of the scapula, the note is occasionally audible upon auscultation of the anterior surface of the thorax above the level of a pleural effusion.

The persistence of egophony in connection with pleural effusions is very variable. When an effusion of medium grade undergoes rapid augmentation, the egophony soon disappears; and when an extensive effusion is undergoing resorption, the sign appears with the gradual diminution in the fluid.

The cause of the sound is a moderate compression of the bronchi due to pulmonary compression, a compression which leads to partial stenosis, yet to a stenosis which can readily be overcome by the force of the vocal vibrations (Eichhorst). Hence, the sign disappears whenever the compression becomes too strong, as well as when the compression has been removed.

Commonly encountered in connection with pleurisy with effusion, egophony is encountered with less constancy over areas of pulmonary collapse and in pulmonary compression in the presence of the consolidation of lobar pneumonia.

Amphoric Vocal Resonance; (Amphorophony; Cavernous Voice).—In the presence of a large superficial pulmonary cavity with bronchial communication or of pneumothorax with a communicating bronchial fistula, the spoken voice upon auscultation frequently has imparted to it an echoing, metallic quality, the sound prolonging itself beyond the emission of the spoken word. The phenomenon is analogous to the amphoric character of the respiratory murmur and of the amphoric percussion sound under similar circumstances and obeys the same physical laws which govern the production of the peculiar quality of the sounds in these instances. The echoing, cavernous quality of the spoken voice under these circumstances constitutes amphoric vocal resonance, amphorophony, or the cavernous voice. When elicited, it constitutes a reliable sign of a cavity containing an air column which vibrates simultaneously with the waves reaching it from the vocal cords.

ADVENTITIOUS SOUNDS

New, superadded, or adventitious sounds, which are engendered in pathologic states of the organs of respiration, may originate in the larynx, trachea, bronchi, lungs, or in the pleural cavity. They comprise various types of râles, the metallic tinkle, the succussion sound, the lung-fistula sound, and the pleural friction sound.

RÂLES

Râles are adventitious sounds which are generated in the larynx, trachea, bronchi, bronchioles, or pulmonary alveoli, as a result of interference with the free ingress and egress of air during inspiration and expiration. The lesion which is responsible for the production of râles, and which will be studied more in detail in connection with the detailed description of the various types of râles, may consist in a diminution of the lumen of the air passage by compression from without, or by turgescence of the mucous membrane within; it may be an obstacle imposed by the presence of serum, mucus, pus, or blood within the bronchial tubes; or it may consist in the cohesion of the walls of the terminal bronchioles and alveoli, which are glued together by a coating of tenacious secretion or fibrinous exudate.

Râles are classified primarily as *dry râles* and *moist râles,* depending upon whether or not moisture is, in the opinion of the examiner, the essential factor in their production.

Dry râles, or **rhonchi,** which are invariably signs of stenosis, are produced by an obstacle to the free ingress and egress of air during respiration in the form of varying degrees of stenosis of the air passages, whether these passages be narrowed by the presence upon their walls of accumulations of tenacious secretion; whether the bronchial lumen be diminished by turgescent swelling of the mucous membrane; or whether the constriction be due to external pressure exerted upon the bronchial tube by bands of adhesions or enlarged thoracic viscera. The generation of the sound in each instance is caused by the passage of the inspired or expired air through a bronchial stenosis into a wider portion of the tube beyond, with the consequent production of aerial whorls. The quality of the râle is dependent for this reason upon the size of the bronchus in which it is generated and the diameter of the constriction which is the causative agent.

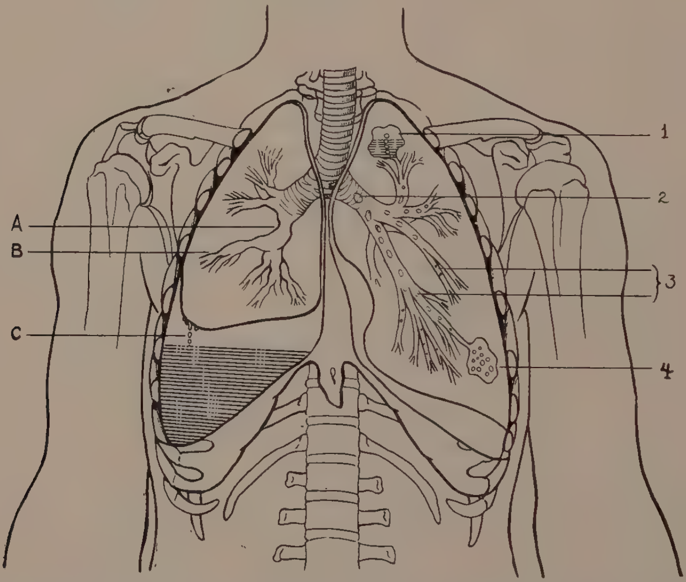


Fig. 216.—Illustrating the physical basis of pathologic physical signs upon auscultation of the thorax. *A,* Sonorous râles; *B,* sibilant râles; *C,* metallic tinkle, or succussion sound; *1* gurgling or bubbling râles; *2,* coarse or mucous râles; *3,* fine or subcrepitant râles; *4,* crepitant râles.

Upon the acoustic influences of these two factors, it is possible to subdivide dry râles into *sonorous râles,* of low pitch, and snoring, often musical quality; and *sibilant râles,* of high pitch, and hissing, whistling, or squeaking quality. Now, since only a very moderate turgescence of the mucous membrane of the smaller bronchi and bronchioles, or the presence of even minimal amounts of secretion upon the walls of these tubes produces considerable stenosis in these small passages, and as their caliber is so limited that the distal portion of the tube cannot act in any considerable degree as a resonating chamber for the aerial whorls which are called into being by the passage of the air through the constricted portion, sibilant râles are practically confined to the distribution of the smaller bronchi and bronchioles, while sonorous râles are produced only in the larger bronchi, the trachea, and the larynx.

Dry râles are most frequently audible during inspiration; less frequently they are audible during inspiration and expiration; and very infrequently indeed are they audible during expiration alone. Dry râles, and this is true of moist râles as well, are frequently audible at some distance from the region of the thorax which overlies the site of their generation; but they always present a point of maximum intensity, which is situated over the portion of the bronchial system in which they are produced.

When dry râles are encountered universally over the thorax, as is the case in bronchial asthma, it is commonly observed that sonorous râles are audible in the main during the first half of inspiration; whereas the sibilant râles come to the fore during the latter half of this act, as a result of the progressive penetration of the inspired air to the ultimate ramifications of the bronchial system. If the râles are audible also during expiration, the opposite sequence of events is observed.

The preponderance of dry râles during inspiration and their frequent absence during expiration is doubtless in intimate relation with the rapidity of the aerial current through the bronchial system during the two phases of the respiratory cycle. In order that whorls of sufficient intensity may be produced in the air passages to produce sound, it is essential that the air pass the stenosis with a fair degree of rapidity, a physical state which obtains during inspiration to a much greater degree than it is present during expiration. This assumption is further substantiated by the fact that dry râles are frequently inaudible during tranquil respiration, to come out clearly during deep inspirations upon the part of the patient.

The relative pitch of dry râles is very readily appreciated even by the inexperienced examiner, the high-pitched, squeaking, whistling sibilant râles contrasting markedly with the deep snoring of the sonorous râles. This variation in pitch possesses in this manner a distinct localizing value in diagnosis.

The intensity of dry râles varies with the force of the respiratory movements, with the degree of stenosis which engenders them, with the site of their production in relation to the thoracic wall, and with the thickness of this wall. When dry râles of considerable initial intensity are generated in the depth of the lung, where they are covered by thick masses of aerated pulmonary parenchyma, the intensity is greatly masked in the transmission of the râles to the thoracic surface. When, on the contrary, râles are generated in bronchial tubes of more superficial distribution, their intensity is as much greater upon auscultation, and they are frequently attended by rhonchal fremitus upon palpation.

Dry râles assume a special intensity and a distinct change in quality when they are engendered in a bronchus which terminates in portions of the lung which are the seat of compression and collapse, and when the bronchus terminates in a pulmonary cavity situated in the periphery of the lung. In this instance the pulmonary excavation acts as a resonating chamber for the bronchial vibrations, with the production of the *consonating râles* of Skoda. The râles in this case are attended by blowing, bronchial breathing and amphoric resonance upon percussion over the cavity.

Dry râles are audible in the earliest stages of bronchial inflammation as a result of moderate turgescence of the bronchial mucous membrane, prior to the pouring out of secretion into the lumina of the tubes, and in the paroxysm of bronchial asthma as a result of constriction of the bronchioles. The *bruit de drapeau* is a dry râle which is audible in certain cases of fibrinous bronchitis, and which is produced by the flapping back and forth of a fragment of adherent exudate or detached mucosa during inspiration and expiration. Sonorous râles are generated in the trachea in the presence of extreme compression of this tube by aneurysm, mediastinal tumor, or enlarged mediastinal glands, as well as in cicatricial stenosis of this tube. Similar râles occasionally arise in the larynx as a result of laryngeal diphtheria and in edema of the glottis.

Moist râles, produced by the passage of air through serum, mucus, pus, or blood in the larger bronchi, or by separation of the walls of the terminal bronchioles and alveoli, which have become adherent as a result of the presence of tenacious exudate, comprise the *crepitant râle,* the *subcrepitant râle,* the *mucous râle,* and the *gurgling râle.*

Crepitant Râle.—The crepitant râle is produced by the separation of the walls of the alveoli, which have become glued together by tenacious secretion during the expiratory recession of the lung. Hence, the râle is audible toward the termination of inspiration.

The crepitant râle is the finest of all râles. Its quality has been compared by Laënnec to the series of sharp cracklings which ensue upon throwing a pinch of salt upon a heated stove. Williams compares the quality of the râle to the sound which is produced upon rolling a lock of hair between the thumb and finger when held near the ear. Both comparisons are inexact, as moisture is an essential element in the production of the crepitant râle. The quality of the râle is aptly imitated upon moistening the tips of the thumb and forefinger, pressing them together, and separating them while the hand is held near the ear. The quality of the crepitant râle is readily confused with the pleural friction sound, when the latter possesses a minor intensity. The differential points between the two sounds are considered in connection with the pleural friction sound in a subsequent paragraph.

The crepitant râle is not infrequently audible upon auscultation of the pulmonary bases in the normal subject who has passed a night breathing tranquilly in the dorsal decubitus. In this instance, however, a few deep inspirations serve to fully inflate the alveoli and to abolish the râle in this area. As this râle is dependent upon a wet lung for its production, it is natural that in the diseases in which it is present it is encountered over the bases in the majority of cases. In all cases in which crepitant râles are encountered over the apices of the lungs, the prognostic significance is grave; and if the râles are localized in this area to the exclusion of other portions of the lungs, it is very suggestive of phthisis as the underlying lesion.

The crepitant râle is detected during the first stage of fibrinous pneumonia, constituting the *crepitus indux* of this disease. It is also audible over the bases of the lungs in the presence of inflammation of the mucous membrane of the terminal bronchioles and pulmonary alveoli in catarrhal pneumonia or capillary bronchitis; and it is audible in the same regions in the presence of pulmonary edema, hemorrhagic infarction of the lung and in partial atelectasis. During the evolution of chronic ulcerative phthisis, and with less constancy in the stage of incomplete consolidation in fibrinous pneumonia, there is occasionally audible a fine, high-pitched crepitant râle, the *mucous click.* As in the case of all crepitant râles, the mucous click is elicited toward the completion of inspiration, when the inspired air has attained the smaller ramifications of the bronchial system and the alveoli have been distended to the maximum by the increased negative pressure in the pleural cavity.

Subcrepitant Râle.—The subcrepitant râle is a moist râle, a trifle coarser in quality than is the crepitant râle. It is produced by separation of the walls of the terminal bronchioles which have become adherent as a result of a coating of tenacious secretion or exudate; hence, it is audible during both inspiration and expiration. The subcrepitant râle occurs during the period of resolution of fibrinous pneumonia, in which disease it constitutes the *crepitus redux;* during the evolution of acute and chronic catarrhal bronchitis; and in the presence of bronchial inflammation in catarrhal pneumonia, pulmonary infarction, and pulmonary edema.

Mucous Râle.—Mucous râles are generated in the larger bronchi and in pulmonary cavities. They are audible during inspiration and expiration; and in quality they present a series of bubbling sounds, analogous to that which is produced by a fluid undergoing rapid fermentation, or the passage of bubbles of air through liquid contained in a small vessel. Their intensity depends upon the quantity of fluid secretion which is present, upon the size of the bronchus or cavity in which they are produced, upon the energy of the respiratory movements, and upon the location of their site of production with reference to the thoracic surface. Mucous râles are most frequently encountered in the evolution of chronic ulcerative phthisis; less frequently in the presence of acute and chronic catarrhal bronchitis, and in the later stages of the paroxysm of bronchial asthma.

Gurgling râles are mucous râles which are generated in the presence of a pulmonary cavity containing fluid, and with a free bronchial communication, which is situated below the level of the fluid. The râles are manifested by a series of gurgling, bubbling sounds which make their appearance toward the completion of inspiration, or merely upon change of posture of the patient. They are due to the passage of the inspired air through the fluid contained in the excavation, and are to be encountered during the advanced stages of chronic ulcerative phthisis associated with cavitation.

As moist râles depend upon moisture of the lung for their production, their characteristics are readily influenced by the depth of the respiratory movements and by the act of coughing, factors which cause shifting of the fluid elements which call the râles into being. Following a particularly deep inspiration or a violent fit of coughing, it is not infrequent for moist râles to disappear transiently, only to reappear with the further accumulation of the pulmonary secretions.

In the interpretation of râles the examiner should take into consideration the number, the size, the uniformity, the time of appearance, the intensity, and the quality of the râles, and whether or not they are consonating.

The *number* of moist râles which may be encountered over the thorax is variable in different instances. In one case there may be only a few râles, well circumscribed to one region of the thorax, and which are only brought out upon deep inspiration. If these discrete râles appear at the termination of inspiration over the bases posteriorly, they frequently disappear after a few inspirations. In this event they are merely crepitant râles which are generated by the full inflation of alveoli which have remained closed during tranquil respiration, and are devoid of pathologic significance. A limited number of moist râles, encountered over the apical portion of the thorax, on the contrary, is of grave prognostic significance, if they are persistent and are encountered in the same area upon consecutive examinations. Circumscribed moist râles in this region are very suggestive of phthisis; but Rosenbach has directed attention to the extreme importance of not mistaking muscular sounds in this region for moist râles, particularly in subjects with powerfully developed pectoral muscles.

Again, as in the case of acute and chronic catarrhal bronchitis and bronchial asthma, moist râles are distributed universally over both sides of the thorax, and are frequently in the latter disease of such intensity as to be audible without the aid of the stethoscope.

The number of the râles is largely dependent upon the amount of the pathologic secretion which is present in the bronchial system, upon the energy of the respiratory movements, and upon the site of the causative lesion with reference to the thoracic surface. When the bronchial secretions are unduly abundant and when the respiratory action is very pronounced, if the lesion is deeply seated in the central portion of the lung, there will be a minimal number of râles demonstrable on account of the interference with their conduction which is offered by the intervening areas of aerated pulmonary tissue.

It occasionally happens that in a given case moist râles are so abundant that the character of the respiratory sounds are determined with difficulty, as the quality of the respiratory murmur is effectually masked by the predominance of the râles. In such event Lasègue recommends auscultation of the respiratory murmur immediately following vigorous coughing efforts upon the part of the patient, at which time the râles are temporarily in abeyance and the quality of the respiratory murmur may be studied.

The *size* of râles is dependent upon the character of the bronchial secretions, the energy of the respiratory movements, and the site of the lesions with reference to the thoracic wall. In respect to their size, râles are divided into large râles, medium râles, and small râles. Large and medium râles are only encountered in the presence of fluid secretions in the lumina of the larger bronchi, while small râles are engendered by the presence of fluid in the bronchi of smaller caliber. In the case of the large and medium-sized mucous râles, the intensity of the râles is in direct proportion to the energy of the respiratory movements and the proximity of the bronchus in question to the thoracic wall.

The *uniformity* of the râles which are encountered should be studied in every case in which moist râles are encountered. In this manner the student determines whether he is dealing with only one type of râle or with various types of râles, and from this information he deduces conclusions as to the portion of the bronchopulmonary system which is at fault. Even in the case of râles of the same character it is occasionally possible to detect differences in the quality and pitch of the râles. Thus, Laënnec held that the crepitant râle of lobar pneumonia is uniformly more intense and of higher pitch than is the crepitant râle of pulmonary edema.

The *time of appearance* of râles with reference to the events of the respiratory cycle is of aid in determining the character of râle with which the examiner is confronted, and serves as a basis for conclusions as to the site of the lesion of the bronchial system.

Thus, the crepitant râle and the great majority of gurgling râles become apparent toward the completion of inspiration; whereas the subcrepitant râle is demonstrable during both inspiration and expiration. Similarly, the mucous râle generated in the larger bronchial tubes is audible during both phases of the respiratory cycle. The study of the time of appearance of râles should be abetted by the careful study of the quality of the râles in question.

The *intensity* of râles depends upon the site of their production, the number of the râles, the size of the râles, and the state of the superimposed pulmonary parenchyma. Râles which are developed in the superficial bronchi are naturally more intense than are râles developing in bronchi of the central portions of the lungs. However, the intensity of râles developing in the depths of the lungs is influenced by the state of the pulmonary parenchyma which is superjacent to these bronchial tubes, and as this is consolidated, compressed, or excavated by phthisis, they assume an undue intensity and special qualities which will be described in a subsequent paragraph. When râles are very abundant and universally distributed throughout the lungs, their intensity is augmented by their number alone, not infrequently to the extent that they are audible without the aid of the stethoscope or, indeed, at some distance from the patient. The size of the râle exerts a perceptible influence upon its intensity. The large and medium râles of the larger bronchi possess a greater intensity than do the small râles which are generated in the smaller bronchial tubes.

The *quality* of the moist râle is more difficult to seize and to define than in the case of dry râles. However, it is upon this difference in quality between dry and moist râles that the examiner bases his conclusions as to whether he is dealing with a dry or with a wet lung. The bubbling quality of gurgling râles is readily appreciated and the incidence of the crepitant and subcrepitant râle at different periods of the respiratory cycle serves as a check in some measure upon these râles.

Moreover, when moist râles are generated in pulmonary tissue which is in close proximity to pulmonary excavations, or not infrequently even when the stomach or colon is excessively distended, the râles take on a musical quality which is closely akin to the quality of the consonating râles of Skoda.

Consonating moist râles possess the musical quality to a marked degree; and, as either pulmonary excavation with bronchial communication or pulmonary relaxation and collapse, furnish excellent conducting media, these râles are transmitted to the surface of the thorax with undue intensity. These râles are attended by bronchial breathing, exaggeration of vocal fremitus and vocal resonance, and by the amphoric percussion sound.

THE METALLIC TINKLE (FALLING-DROP SOUND)

During auscultation of the thorax which is the seat of hydro-hemo- or pyo-pneumothorax, and with less constancy during auscultation over a large pulmonary cavity which contains fluid, a sound is occasionally audible during inspiration and expiration which resembles that which is produced by drops of water falling from a height upon the surface of water contained in a cistern. The sound has a hollow, echoing, metallic quality; it is most frequently present during inspiration, though occasionally heard during expiration as well; and it is frequently brought out by changes of posture, speaking, coughing, or by jarring or shaking the subject.

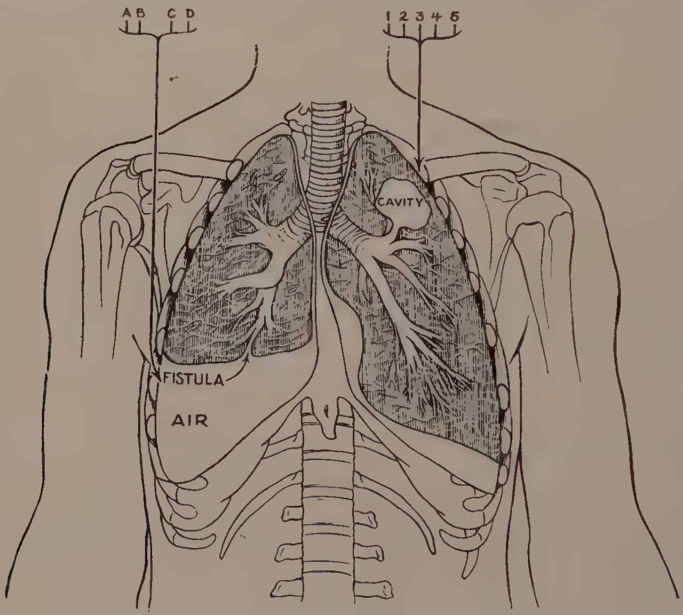


Fig. 217.—Physical basis of pathologic physical signs upon percussion and auscultation of the thorax.

A, cracked-pot sound; *B*, Biermer's phenomenon; *C*, pectoriloquy; *D*, lung-fistula sound; *1*, cracked-pot sound; *2*, Wintrich's change of sound; *3*, Friedreich's change of sound; *4*, bronchial or amphoric respiration; *5*, whispering pectoriloquy.

The physical genesis of the falling-drop sound is obscure, and several theories have been evolved in the attempt to explain the mode of production of the sound in the various pathologic cases in which it has been encountered. Laënnec originally attributed the sound to the dripping of fluid from the retracted inferior border of the lung to the surface of an accumulation of fluid occupying the lower portion of the pleural cavity. Baas opposed this theory, asserting that droplets of fluid which are formed upon the superior walls of a cavity have a tendency to glide along the walls of the cavity, instead of falling abruptly from these superior regions. Leichtenstern, however, encountered a very pure falling-drop sound in a case of pyo-pneumothorax, which was only demonstrable when the patient passed from the dorsal decubitus to the sitting posture. Upon autopsy the case presented villosities of the pleural surface, which were immersed in the fluid with the patient in the dorsal decubitus, and which dripped fluid when the patient was placed in the upright posture.

Another theory of the mode of production of the metallic tinkle is the assumption of the bursting of bubbles which are formed upon the surface of an accumulation of fluid in the pleural cavity. Force is lent to this explanation in certain cases by the fact that the sound is elicited immediately following the succussion sound upon jarring the subject.

Debove and Trémolières find a striking similarity between the quality of the falling-drop sound and that of the Hippocratic succussion sound when the latter is elicited by gentle agitation of the patient. These authors believe that the metallic tinkle is due neither to the dripping of fluid from the retracted borders of the lung nor to the bursting of bubbles upon the surface of the pleural fluid; but that it is due to the generation of a light wave upon the surface of the fluid enclosed in the pleural cavity, the latter acting as a resonating chamber for the slight sound which is so generated. They hold that the rhythmic diaphragmatic movements are alone sufficient to call into being the causative waves in the intrapleural fluid.

It has also been asserted that the metallic tinkle is only generated in connection with accumulations of fluid in the pleural cavity when there is a patent bronchial communication with the pneumothorax; and that the sound is to be attributed to the bursting of a bubble which has formed at the orifice of the bronchopulmonary fistula at the moment of full inspiration.

HIPPOCRATIC SUCCUSSION (SPLASHING SOUND)

In the presence of hydro-, hemo- or pyo-pneumothorax, when the upper portion of the trunk is abruptly jarred or shaken, the ear of the examiner meanwhile being applied closely to the thoracic wall, a distinct sound of splashing is frequently audible, which is analogous to that which is produced by suddenly moving a partially filled cask. The succussion sound is a reliable sign of the presence of air and fluid in the pleural cavity; hence it is absent in pleurisy with effusion. The succussion sound arising within the pleural cavity should not be confused with similar splashing sounds which are generated within a dilated stomach.

While in the case of large pulmonary cavities containing air and fluid the physical conditions are ideal for the generation of Hippocratic succussion, it is rare that these excavations are so favorably situated with reference to the thoracic surface that succussion sounds are appreciable over them.

THE PLEURAL FRICTION SOUND

In the normal subject, as a result of the smooth, polished surface of the visceral and the parietal pleura, moistened by a moderate amount of serous fluid, these membranes glide noiselessly over each other during the respiratory movements. During inflammation of the membrane, however, and as a result of the excessive extraction of the body fluids which accompanies prolonged diarrhea and profuse hemorrhage, as well as in the presence of miliary tuberculosis and carcinomatous infiltration of the pleura, there is generated a pleural friction sound, which is audible upon auscultation of the surface of the thorax. Jürgensen and Waldenburg would make a distinction between pleuritic friction, which is due to inflammatory processes of the pleural membrane, and pleural friction, which occurs with roughening of the membrane by the nodules of acute miliary tuberculosis, carcinomatous infiltration of the pleura, and osseous excrescences of the ribs, which they do not regard as essentially inflammatory in origin. As the pleural membrane is extremely prone to undergo inflammatory changes in the presence of even minor grades of irritation, it is not improbable that all of these conditions are attended by pleuritis of varying intensity.

The pleural friction sound presents extensive variations in its quality and intensity. In cases of slight pleural involvement it possesses a very light, grazing sound, analagous to that which is produced when the finger lightly strokes a silken fabric. In the presence of more severe pleural inflammation, the sound is very similar to that which is produced upon walking upon dry, new-fallen snow, or to the creaking of a new leathern saddle.

While in many instances the pleural friction sound is a continuous sound, appreciated throughout the greater duration of the respiratory phase in which it is audible, in other cases it presents one or more interruptions, as if the roughened pleura were successively surmounting obstacles during the excursion of the lung.

The intensity of the pleural friction sound is in direct proportion to the intensity of the respiratory movements, to the extent of the pleura which is diseased, and to the severity of the inflammatory process. In cases of very limited pleural involvement the sound is very faint, requiring a trained ear for its detection. In other cases the sound is so intense as to be audible without the aid of the stethoscope, and is accompanied by distinct pleural friction fremitus upon palpation. Voluntary acceleration of the respiratory rate and increased depth of inspiration serve to increase the intensity of the sound. The intensity of the friction sound may be artificially increased by the exercise of pressure in the intercostal spaces over the area in which it is encountered. Holding the breath at the end of inspiration causes the pleural surfaces to remain in apposition. The respiratory cycles immediately follow-

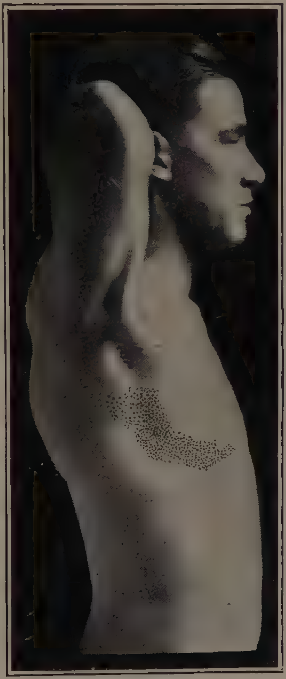


Fig. 218.—Usual site of pleural friction sound.

ing this procedure may be accompanied by friction sounds which have hitherto been inaudible. Holding the breath at the end of inspiration and rapidly raising the arm above the head may cause a reverse movement of the parietal upon the visceral pleura, thus eliciting a friction sound otherwise inaudible. Occasionally the friction sound is transitorily suppressed after several deep inspirations, probably as the result of smoothing out of the rugosities of the membrane, as the sound reappears upon resuming respiration after its temporary suspension.

Pleural friction is most frequently audible during inspiration, with less frequency during inspiration and expiration, and very rarely indeed is it audible during expiration alone.

The localization of pleural friction naturally corresponds with the distribution of the inflammatory disease of the pleura. It is encountered most frequently in the lower axillary and infra-axillary regions, where it is most frequently observed along an ascending and descending plane. When, in these areas, the friction seems to occur in a transverse direction, it is suggestive of the limitation of the vertical excursion of the lung by pleural adhesions. Pleural friction is not frequently encountered over the apical portions of the lungs, as these regions enjoy only a limited range of mobility during the respiratory movements; but when pleural friction is encountered here, it is very suggestive of tuberculous lesions which are complicated by a fibrinous pleurisy.

The duration and persistence of pleural friction are subject to wide variations. During the course of serofibrinous pleurisy the friction sound appears prior to the development of the effusion; it disappears with the advent of the effusion; and not infrequently it again becomes audible with resorption of the effusion, occasionally to persist thereafter during the entire life of the individual. In cases of abortive acute fibrinous pleurisy, the sound may be present upon one examination, to disappear immediately thereafter. When pleural friction develops in the supra- and infraclavicular regions in connection with chronic ulcerative phthisis, the sounds commonly persist throughout the course of the disease.

As pleural inflammation very frequently complicates diseases of the lungs which are attended by râles, it is natural that pleural friction and râles of various types are frequently present in the same subject; and in many instances their differential diagnosis is attended by some difficulty. In this connection it is to be recalled that upon exerting pressure with the stethoscope in the intercostal spaces the intensity of pleural friction is commonly accentuated, while this maneuver is without influence on the intensity of râles.

Moreover, râles are more continuous than is the case with pleural friction; râles change in character or transiently disappear after coughing attacks; and compression of the thorax in the presence of pleural disease is acutely painful, while similar compression of the chest in pulmonary disease unattended by pleurisy is provocative of only slight pain if indeed there is any pain.

THE LUNG-FISTULA SOUND

In the presence of hydropneumothorax with a bronchial communication opening into the pleural cavity below the level of the fluid, Riegel first described the lung-fistula sound. The sound is manifested by a series of bubbling, gurgling sounds upon auscultation of the diseased side under suitable conditions. When, under proper maneuvers, the aerial content of the pleural cavity is rarefied in the region superjacent to the fluid, during inspiration a portion of the inspired air enters the cavity below the level of the fluid, and in ascending to the surface, engenders bubbles which produce the gurgling, lung-fistula sound. Unverricht noted the sound following partial aspiration of a hydropneumothorax, the superjacent air in this instance being rarefied by the evacuation of the fluid.

In Riegel's case the sound appeared without aspiration of the fluid. In this case, as soon as the patient was placed in the sitting posture, he expectorated a considerable quantity of the pleural fluid, which passed by way of the bronchial fistula. Following this partial evacuation of the fluid, the balance between the aerial pressure in the bronchial system and in the pleural cavity was disturbed; and during inspiration a portion of the inspired air entered the pleural cavity below the level of the fluid, and in its ascent through this latter engendered the lung-fistula sound.

The sound may be elicited without aspiration or expectoration of the fluid by the procedure of Meezenbrock. This author has the patient assume the lateral decubitus, lying upon the side of the disease. He then seizes the dependent side of the thorax between the hands, and by compression evacuates a portion of the pleural fluid into the air passages through the fistulous opening. When the patient assumes the sitting posture and the pressure is gradually released from the diseased side, the air penetrates into the pleural cavity through the fistulous opening and produces the lung-fistula sound.

CHAPTER XXVIII

AUSCULTATION OF THE CIRCULATORY SYSTEM

Object and Technic.—Auscultation is employed in the study of the circulatory organs to determine the intensity, quality and pitch of the sounds of the heart, their rhythm, and the presence or absence of certain adventitious sounds arising in the heart, pericardium and arteries.

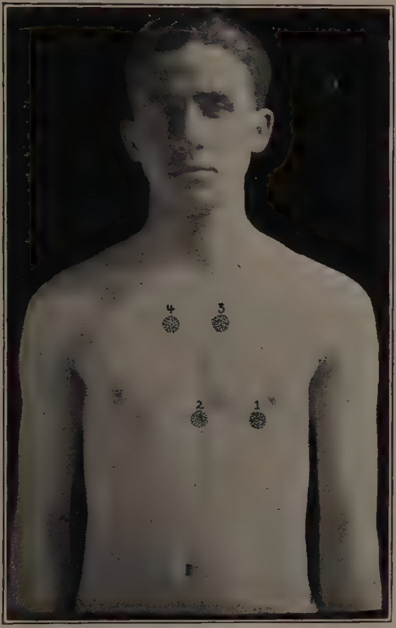
During auscultation of the precordia mediate auscultation is to be employed in preference to immediate auscultation. During the examination no article of wearing apparel should intervene between the integument of the subject and the bell of the stethoscope. The attitude of the subject should be free from muscular tension; and, owing to the influence of the bodily attitude upon certain of the phenomena, the examination should be conducted with the subject in the erect and in the recumbent posture. In a limited number of cases, on account of the urgent dyspnea attending the underlying lesions, the examiner is forced to dispense with the examination in the recumbent posture.

In the examination of the sounds of the heart, auscultation is not practiced over the anatomic sites of the several valves, but in certain definite areas of the thorax in which the different heart sounds are audible with the maximum intensity and purity.

Auscultatory Valve Areas.—Each of the four valves of the heart has a corresponding area upon the thoracic surface at which the sound produced by closure of the valve in question is more distinctly audible than elsewhere. As stated, these areas do not correspond to the points upon the thoracic wall which are nearest to the anatomic sites of the valves.

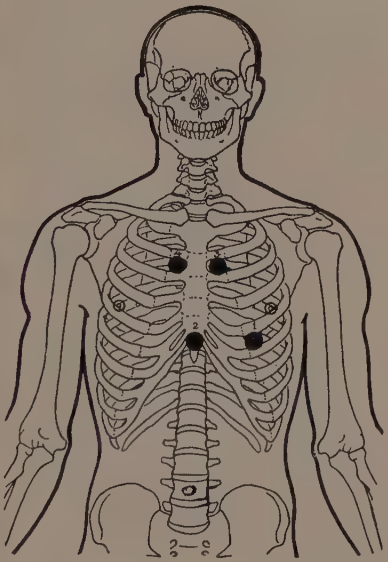
The sound produced by closure of the mitral valve is most clearly audible in the *mitral area,* which is situated in the fifth left intercostal space over the apex of the heart, although the anatomic site of this valve is behind the left half of the sternum at the level of the fourth costal cartilage, the sounds being better transmitted through the left ventricle to a point where it nears the surface of the body than through the intraventricular septum and right ventricle.

The sound produced by closure of the aortic valve is most clearly audible at the *aortic area*, which is situated in the second intercostal space immediately adjacent to the right sternal border, although the anatomic site of this valve is posterior to the left half of the sternum at the level of the third interspace, the sound in this case being better transmitted through the blood stream and the aorta to a point where it first nears the surface than through the numerous tissues lying between the anatomic site of the valve and the chest wall.



A.

Fig. 219-*A*.—Auscultatory valve areas of the heart. *1*, Mitral area; *2*, tricuspid area; *3*, pulmonary area; *4*, aortic area.



B.

Fig. 219-*B*.—Auscultatory valve areas of the heart. *1*, Mitral area; *2*, tricuspid area; *3*, pulmonary area; *4*, aortic area.

The *tricuspid area*, at which sounds arising from the tricuspid valve are most distinctly audible, is situated over the lower portion of the sternum, the anatomic site of this valve being situated behind the right portion of the sternum between the level of the fourth and sixth costal cartilages. Thus, the tricuspid valve lies almost directly beneath this valve area, the sounds being transmitted directly through the right ventricle and chest wall.

The *pulmonary area*, at which sounds generated by the action of the pulmonary valve are most distinctly audible, occupies a point immediately to the left of the sternum in the second intercostal space, the anatomic site of this valve being posterior to the junction of the third left costal cartilage with the sternum. The sounds, as in the case of those produced at the aortic valve, are better transmitted through the pulmonary artery and blood stream than through the numerous tissues overlying the pulmonary valve.

The Normal Heart Sounds.—The heart in its rhythmic action produces two sounds, which are termed respectively the *first* and the *second* sound of the heart. The first sound is audible most clearly in the region of the cardiac apex in the normal subject, while the normal second sound is most clearly audible at the base of the heart.

The *first sound* of the heart is audible during ventricular systole; it is lower in pitch and of longer duration than is the second sound; and it has been compared acoustically to the sound of the word "lubb."

The mechanism of production of the first sound of the heart is somewhat obscure. A large element of the sound is produced by the friction of the interlacing bundles of muscle during ventricular contraction, and by the closure of the auriculoventricular valves. Probable but uncertain factors in the production of the sound comprise vibration of the chordæ tendineæ following closure of these valves, the sudden distention of the proximal portions of the aorta and pulmonary artery, and, in certain instances, the impact of the cardiac apex against the thoracic wall. The sound is, however, a combined muscular and valvular sound, and a careful study of the two essential elements of the sound is of service in drawing conclusions as to the integrity of the myocardium.

The *second sound* of the heart is audible during the early portion of ventricular diastole. The sound marks the beginning of diastole; but it consumes only the early portion of diastole, hence is really protodiastolic in time. The second sound is of higher pitch and distinctly shorter duration than is the systolic sound; and it has been likened acoustically to the sound of the word "dup." The second sound of the heart is produced by the sudden coaptation of the segments of the semilunar valves, which guard the orifices of the aorta and the pulmonary artery.

Occasionally there is to be detected a *third sound* of the heart, occurring approximately 0.13 second after the commencement of the second heart sound. Gibson and Thayer attribute the third sound to sudden tension of the cusps of the mitral valve, occurring with the inrush of the blood at the commencement of diastole. Einthoven, on the other hand, attributes the sound to after-vibrations of the cusps of the aortic valve. Other investigators have attributed the third heart sound to forcible auricular systole. The precise mode of generation of the sound is thus not definitely established. It is most frequently to be detected in children during periods of bradycardia with the subject in the left lateral decubitus.

Intensity of the Heart Sounds.—Upon auscultation of the precordia of the normal subject, it is noted that the individual sounds arising at the different valve areas of the heart are not of uniform intensity. Thus, although the first sound of the heart is produced by the combined action of the ventricles and the auriculoventricular valves, it is observed that the first sound at the mitral area is lower in pitch and of somewhat greater duration than is the tricuspid first sound. Similarly, in the examination of the component valves concerned in the production of the second sound, it is observed that in the adult subject the second sound at the aortic area is of greater intensity than is the second sound generated by closure of the pulmonary valve; whereas, in the child the condition is reversed, the pulmonary sound exceeding in intensity the aortic second sound.

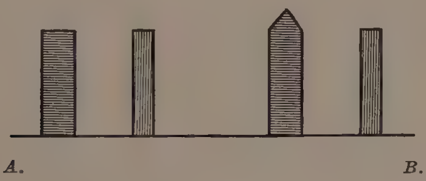


Fig. 220.—*A*, Normal first and second sounds. *B*, Accentuated first sound.

VARIATIONS OF INTENSITY

The intensity of the cardiac sounds, as elicited upon auscultation of the several valve areas, varies in normal subjects of different types, with variations in the thickness of the thoracic wall, with the state of the subjacent pulmonary tissues, and with endocardial and exocardial disease. A correct evaluation of the intensity of the heart sounds can only be made after extensive clinical experience. Variations in intensity may involve one or both sounds of the heart, and the change in intensity frequently is associated with a definite change in the quality and pitch of the sound.

Accentuation of Both Sounds.—Both first and second sounds of the heart exhibit accentuation following the ingestion of stimulants, and during physical effort or emotional excitement. The accentuation in this instance is transient, and is devoid of pathologic significance. A more permanent accentuation occurs with cardiac hypertrophy, during exophthalmic goiter, and in the presence of acute febrile diseases.

An apparent accentuation of both cardiac sounds is occasionally encountered in subjects with very thin chest walls, and in patients in whom fibroid retraction of the anterior border of the left lung exposes the heart freely to the anterior thoracic wall. Similarly, consolidation or carcinomatous infiltration of the lappet of lung overlying the heart transmits the normal cardiac sounds to the surface of the thorax with undue intensity, simulating a true accentuation of the tones.

Diminished Intensity of Both Sounds.—In the robust subject with a deep thorax, and in the obese subject with thick subcutaneous tissues, there is a pseudo-diminution of the intensity of the heart sounds upon auscultation of the precordia. Similarly, in hypertrophic emphysema, when the distended anterior borders of the lungs intervene between the heart and thoracic wall, the cardiac sounds are enfeebled without possessing any reference to the state of the myocardium. In the presence of moderate pericardial effusion, when a limited amount of fluid intervenes between the heart and the chest wall, there is noted a diminution in the intensity of both sounds.

In the presence of myocardial degeneration and cardiac dilatation, however, the cardiac sounds are enfeebled as a result of impairment of the integrity of the myocardium. General asthenia from chronic wasting disease is likewise attended by diminution of the intensity of the heart sounds.

Accentuation of the First Sound.—An increase in the intensity of the first sound of the heart at the mitral area, of moderately increased duration, followed by an accentuated aortic sound, is indicative of left ventricular hypertrophy. When cardiac dilatation is imminent, the first sound as elicited at the apex is loud, but is of brief duration and has engrafted upon it the valvular quality of the normal second sound of the heart.

During the course of acute fevers, when the ventricle is contracting powerfully in the presence of low arterial tension, the first sound of the heart at the apex is accentuated. In exophthalmic goiter the first sound is likewise increased in intensity, but with a diminution in the muscular element and an increase in the valvular quality of the sound.

Diminution of the First Sound.—Myocardial degeneration and cardiac dilatation are attended by diminution in the intensity of the first sound at the apex. Similarly, the asthenia of chronic wasting disease, anemia, and prolonged fever results in diminution in the first sound, the tone acquiring a valvular quality analogous to that of the normal second sound of the heart.

Accentuation of the Aortic Sound.—The aortic sound is accentuated in all physical states which are attended by increased tension in the greater circulation. Hence, it is encountered in cases of angiospasm due to transient vasoconstriction, in arteriosclerosis, chronic nephritis, uremia, and in apoplexy. When the proximal portion of the aorta and the semilunar valves participate in general arteriosclerosis, the aortic sound is accentuated, metallic and ringing. Pregnancy is attended by moderate accentuation of the aortic sound of the heart.

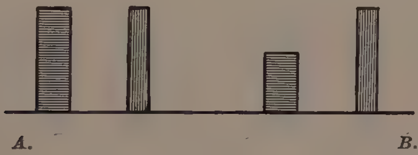


Fig. 221.—*A*, Normal first and second sounds. *B*, Diminished first sound.

In left ventricular hypertrophy arising as a sequence of arteriosclerosis or chronic hypertension in the greater circulation, there is coincident accentuation of the aortic sound and of the first sound at the apex. With the inception of cardiac dilatation under these circumstances the first sound at the apex becomes progressively enfeebled, with maintenance of the accentuation of the aortic sound. Such a change in the relative intensity of the two sounds is always of grave prognostic import.

Diminution of the Aortic Sound.—Enfeeblement of the aortic sound accompanies lowering of the blood pressure in the greater circulation incident to profuse hemorrhage, anemia, relaxation of the peripheral arterioles, and when, as a result of mitral or aortic stenosis or insufficiency, a diminished quantity of blood is ejected into the aorta during ventricular systole. The aortic sound is likewise diminished in intensity in the presence of myocardial degeneration and dilatation of the left ventricle, unattended by hypertension in the greater circulation.

Accentuation of the Pulmonary Sound.—Hypertension in the pulmonary circulation is attended by accentuation of the pulmonary sound of the heart. Such hypertension may be of endocardial or of exocardial origin. Regurgitant and stenotic lesions at the mitral or aortic valve, by causing the blood to accumulate in the pulmonary circuit, result in accentuation of the pulmonary sound.

Pulmonary hypertension of exocardial origin occurs in the presence of obstructive pulmonary disease, and in mechanical congestion of the lungs induced by the pressure of tumors or enlarged glands upon the great veins returning blood from the lungs to the left auricle. Accentuation of the sound due to pulmonary disease is encountered during the course of phthisis, pneumonia, hypertrophic emphysema and cirrhosis of the lung.

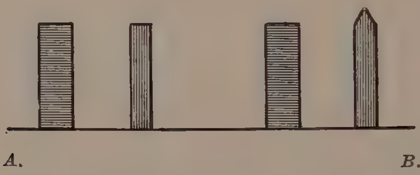


Fig. 222.—*A*, Normal first and second sounds. *B*, Accentuated second sound.

In the presence of right ventricular hypertrophy arising from any cause, associated with integrity of the pulmonary valve, the second sound is accentuated at the pulmonary area.

Diminution of the Pulmonary Sound.—Diminution in the intensity of the pulmonary sound is indicative of imminent failure of the right ventricle. When, during the course of left-sided valvular disease or obstructive disease of the lungs, a pulmonary sound which has shown accentuation becomes enfeebled, it indicates right ventricular dilatation or the incidence of tricuspid regurgitation.

Quality of the Heart Sounds.—The low-pitched, muscular booming quality essentially belongs to the first sound of the heart, whereas the high-pitched, valvular quality is distinctive of the second sound. In the presence of myocardial degeneration, when the ventricular contractions are increased in frequency, there is a diminution in the muscular element of the first sound at the apex, the sound acquiring a valvular quality analogous to that of the normal second sound. Similarly, in exophthalmic goiter there is noted a diminution in the muscular element and an assumption of the valvular quality by the first sound at the apex.

When arterial sclerosis involves the proximal portion of the aorta and the semilunar valves, the second sound of the heart at the aortic area has imparted to it a loud, metallic and ringing quality, quite dissimilar to the normal second sound. Similarly, in the presence of extensive pneumothorax or gastric distention, and when an extensive pulmonary excavation adjacent to the heart contains air, the cardiac sounds have a metallic quality of exocardial origin imparted to them.

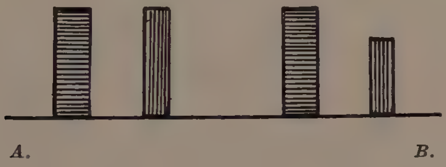


Fig. 223.—*A*, Normal first and second sounds. *B*, Diminished second sound.

Pitch of the Heart Sounds.—During the course of acute febrile diseases, and in the presence of acute myocarditis, the first sound of the heart as appreciated at the apex is increased in pitch, the change in pitch being associated with a diminution in the duration of the sound. Increased pitch of the second sound is encountered at the aortic or pulmonary areas when the pressure is raised in the general or pulmonary circulations. Elevation of the pitch of the sounds also attends the alteration in quality which is noted when large air-containing cavities occupy regions of the thorax or abdomen adjacent to the heart.

Duration of the Heart Sounds.—In the presence of hypertension affecting the greater circulation, attended by adequate compensation by the left ventricle, the first sound of the heart at the apex is moderately prolonged, the muscular element of the sound to a great degree obscuring the valvular element.

In the presence of myocardial degeneration, on the contrary, there is noted a diminution in the duration of the first sound of the heart. The first sound under these circumstances is likewise diminished in intensity, is raised in pitch, and has engrafted upon it the valvular quality of the normal second sound of the heart.

REDUPLICATION OF THE HEART SOUNDS

Either the first or the second sound of the heart may under appropriate conditions become doubled or reduplicated. Usually the examiner encounters a reduplication of the first or second sound alone, but occasionally there is to be detected a reduplication of both sounds of the heart.

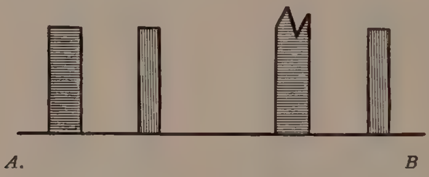


Fig. 224.—*A*, Normal first and second sounds. *B*, Reduplicated first sound.

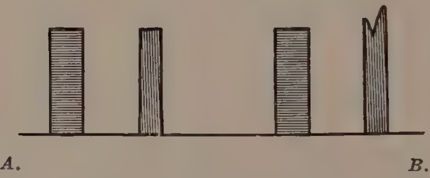


Fig. 225.—*A*, Normal first and second sounds. *B*, Reduplicated and accentuated second sound.

Reduplication of the first sound of the heart is manifested acoustically by a sound resembling the words "lur-rup-dup." An adequate solution of the method of production of the abnormal sound is beset with difficulties. The phenomenon has been ascribed to unequal tension of the leaflets of the mitral and tricuspid valves during ventricular systole. It has likewise been suggested that the phenomenon is caused by tardy contraction of the papillary muscles, as these muscles are supplied by terminal arteries and are readily subject to fatigue.

The sound produced by reduplication of the first sound of the heart is readily confused during a clinical examination with a presystolic mitral murmur when this is followed by a normal or snapping first sound of the heart. The reduplication, which is commonly limited to the apical area has been noted in mitral stenosis, myocardial degeneration, arteriosclerosis, and chronic adhesive pericarditis.

Reduplication of the second sound has been compared acoustically to the sound of the spoken words "lub-durrup." The phenomenon is attributed to asynchronous closure of the aortic and pulmonary valves, occurring as a result of unequal tension in the greater and lesser circulations. Under these circumstances the ventricle which has to overcome the heightened pressure contracts more slowly than its fellow and the semilunar valves close at an appreciably later period than do those of the corresponding valve of the opposite side.

This circulatory imbalance occurs in all states which raise the pulmonary blood pressure, such as hypertrophic emphysema, cirrhosis of the lung, the consolidations of pneumonia and phthisis, and left-sided valvular lesions of the heart. Similarly, the pressure is raised in the general circulation in the presence of arteriosclerosis, temporary angiospasm, and chronic nephritis.

Reduplication of the second sound has occasionally been noted in the normal subject during forced inspiration.

GALLOP RHYTHM

The introduction of a third sound into the cardiac cycle in certain cases of cardiac asthenia and cardiovascular disease constitutes the *gallop rhythm* of Potain. The superadded sound may occur just prior to ventricular systole, or it may be audible during the early portion of ventricular diastole.

Presystolic gallop rhythm is characterized by the occurrence of a third sound which is audible just prior to the first sound of the heart, simulating acoustically the presystolic murmur. It has been noted especially in association with mitral stenosis, and has been ascribed to undue force of the auricular systole. The sound has also been attributed to asynchronous contraction of the right and left ventricles. Aside from mitral stenosis, presystolic gallop rhythm has been noted in association with hypertension in the greater circulation with imminent failure of the left ventricle, and during the asthenia of prolonged infections.

Diastolic gallop rhythm is to be considered merely an exaggeration of the third sound of the heart, and is to be attributed to the sudden tension of the mitral cusps upon the inrush of the auricular blood column during early diastole. Occurring in conditions associated with cardiac asthenia, the later during diastole the sound has its inception, the more grave is the prognosis.

ADVENTITIOUS SOUNDS

In the presence of disease of the circulatory organs various adventitious or new sounds are generated within the chambers of the heart (endocardial), or in the pericardium, lungs, pleura or vessels (exocardial).

Adventitious sounds comprise endocardial murmurs, the cardiorespiratory murmur, pericardial friction, the pericardial succussion sound, and murmurs arising in the arteries and veins.

ENDOCARDIAL MURMURS

Endocardial murmurs are adventitious sounds arising within the chambers of the heart or in the proximal portions of the great arteries springing from the ventricles. They may be superadded to the cardiac sounds, or they may entirely replace the sounds of the heart.

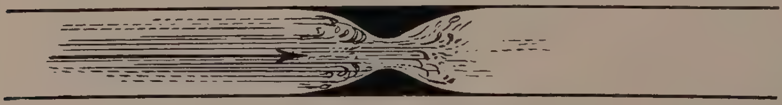


Fig. 226.—Illustrating the physical basis of murmurs generated by diminution of lumen.

The manner in which endocardial murmurs are generated varies in the case of different murmurs, and the mechanism concerned is not always clear. All of the mechanical factors concerned in their production are not established or understood. In the case of stenotic murmurs, which are provoked by diminution of the lumen of an orifice, the physical conditions are in all respects analogous to those which obtain when any circulating fluid passes through a constricted orifice into a wider chamber beyond, in which event vibrations, eddies, or the so-called "fluid veins" are engendered in the circulating fluid with the consequent induction of sound and palpable vibration. As long as the normal quota of normal blood passes through a normal heart with intact endocardium and valves, no sound is engendered save the normal heart sounds. But when the blood stream is forced through a narrowed or stenotic orifice into a wider chamber beyond, or when the blood is permitted by an incompetent valve to regurgitate into a chamber of the heart, an endocardial murmur is prone to become audible upon auscultation of the precordia.

In the presence of dilatation of a ventricle, the muscular ring of the auriculoventricular valve is prone to yield to such a degree that the normal valve cusps are incapable of occluding the abnormally large orifice, resulting in a condition of relative insufficiency or regurgitation and the consequent generation of an endocardial murmur. Similarly, in the case of the aortic and pulmonary valves, dilatation of the proximal portion of the aorta or of the pulmonary artery may so alter the relation of the size of the normal orifice to the arterial caliber as to produce fluid veins in the artery with the production of a murmur in the absence of stenosis of the orifice.

The density of the blood also influences the generation of endocardial murmurs. In the presence of grave anemia the density of the blood is frequently reduced to such a degree that the blood column is whipped into eddies while passing through normal orifices into chambers of normal caliber, producing *hemic* or *functional* murmurs in the absence of disease of the heart.

Additional factors which may be concerned in the generation of endocardial murmurs comprise vibrations of the valvular cusps and chordæ tendineæ, as well as of the walls of the aorta and pulmonary artery.

A certain degree of endocardial pressure is essential to the generation of endocardial murmurs. This is evinced by the fact that endocardial murmurs remain distinctly audible as long as cardiac compensation is maintained, and become indistinct or lost with the supervention of cardiac dilatation.

CHARACTERISTICS OF ENDOCARDIAL MURMURS

Skoda first called attention to the diagnostic importance of the characteristics or properties of endocardial murmurs, and showed that by the detailed study of these several characteristics murmurs arising at the several orifices of the heart may be isolated, and their site of production may be determined.

These properties or attributes comprise the *intensity, point of maximum intensity, line of transmission, time, quality,* and *duration* of the murmurs.

Intensity.—The intensity of a murmur is dependent upon the state of the myocardium, the velocity of the circulating blood stream, the size of the orifice and the dimensions of the cavity of the heart in question, and the density of the blood.

Just as a certain degree of endocardial pressure is essential to the generation of a murmur, so also is the intensity of the murmur a good index to the state of the myocardium. Thus, a loud murmur suggests the presence of cardiac hypertrophy, while a faintly audible murmur is very suggestive of inadequate compensation. Moreover, a change in the intensity of a murmur during daily examinations affords an index to the reserve power of the heart, a change from a loud to a soft, faintly audible murmur suggesting a failing heart, whereas a progressive increase in the intensity of a murmur from day to day is suggestive of cardiac improvement provided the valvular pathology is not increasing as in the case of a progressing endocarditis.

In the case of systolic murmurs, in the generation of which the blood stream is propelled with considerable force by the ventricular contractions, the intensity is usually greater than that of diastolic murmurs, in the generation of which the blood stream is propelled by the elastic recoil of the aorta or pulmonary artery. Similarly, in the case of presystolic murmurs the blood stream is not propelled by auricular systole with the same degree of force as in the case of systolic murmurs engendered during ventricular systole.

In general, the intensity of a murmur is in direct proportion to the size of the orifice at which it is produced and the dimensions of the cavity into which the blood column is propelled; namely, the greater the degree of stenosis, the greater the intensity of the murmur which is generated at the orifice in question. However, in drawing conclusions as to the degree of stenosis from the intensity of the murmur, the examiner should consider its duration and quality, the state of the myocardium, and the extraneous factors which may influence the intensity of endocardial murmurs.

The posture of the patient exerts a striking influence upon the intensity of endocardial murmurs. In general, regurgitant murmurs are most intense when the subject assumes the dorsal decubitus, while stenotic murmurs exhibit their maximum intensity with the patient in the erect posture. The influence of posture upon the intensity of murmurs is exhibited chiefly in the case of murmurs arising at the mitral and tricuspid valves, however, as murmurs generated at the aortic and pulmonary valves are only slightly influenced by these changes.

In the presence of grave anemia an endocardial murmur is apt to exhibit an increase in intensity, and under these circumstances the quality of a murmur is frequently altered.

The intensity of endocardial murmurs is influenced by the depth of inspiration, as well as by the state of the superimposed pulmonary tissues and the chest wall. Upon forced inspiration the anterior borders of the lungs cover an extensive portion of the heart and diminish the intensity of endocardial murmurs, as appreciated upon auscultation of the precordia. Similarly, the voluminous anterior pulmonary borders in hypertrophic emphysema, by intervening between the heart and chest wall, diminish the intensity of sounds emanating from the chambers of the heart. In the robust subject with deep thorax, in the obese subject with thick chest wall, and in the thorax which is the seat of osseous deformity the intensity of endocardial murmurs is masked without reference to the state of the myocardium. The presence of large air-containing cavities adjacent to the heart, on the contrary, and the presence of extreme tympanites are capable of producing a false augmentation in the intensity of endocardial murmurs.

Physical exertion increases the intensity of endocardial murmurs when the heart muscle is of sufficient strength to compensate for the added strain. In the presence of a weak muscle where the added strain is not met by muscular response, the murmur is less easily heard. Judicious exercise frequently reveals the presence of an obscure murmur, which is not detected during examination with the patient in repose. This is true especially when the heart muscle is weak and the valve ring dilates under the added strain. It is also true when there is but slight thickening or roughening of a valve and increased velocity of the blood stream is necessary to produce a vibration.

Point of Maximum Intensity.—Every endocardial murmur has a point of maximum intensity, the point at which it is most distinctly audible upon auscultation of the thorax. These points correspond very accurately, on the whole, with the points at which the closure of the normal valves are most clearly audible; namely, in the four acoustic valve areas. Thus, a murmur which is generated at the mitral valve is usually heard with the greatest intensity at the mitral area; whereas, a murmur which is generated at the pulmonary valve is most clearly audible at the pulmonary area. There are some variations to this rule, mitral murmurs being frequently heard with maximum intensity a little to the left or right of the apex and aortic regurgitant murmurs in the fourth intercostal space to the left of the sternum. The latter is probably due to rotation of the hypertrophied heart.

This selective transmission of the sound in its maximum intensity to the surface of the thorax in the case of the different murmurs is accounted for by the fact that the sound is conducted most readily in the direction of the blood current which called it into being, and by differences in the conductivity of the component portions of the heart and of the thorax.

Line of Transmission.—The majority of endocardial murmurs are audible not only at their points of maximum intensity, but are transmitted thence in directions which vary in the individual murmurs, constituting the line of transmission or area of propagation of the murmur. The direction in which a given murmur is to be transmitted is determined in certain instances by the direction of the blood stream, in other cases by the arrangement of the papillary muscles and ventricular wall, and again by the relations of the several chambers of the heart to the lungs, mediastinal structures and the chest wall.

The distance to which a murmur is transmitted is dependent upon the state of the myocardium, whether hypertrophied or the seat of retrogressive changes, the initial intensity of the murmur, the size of the heart, and the state of the superimposed pulmonary tissues and chest wall.

Thus, with augmentation of the propulsive force of the left ventricle in hypertrophy of this chamber, and the increase in the size of the heart in massive hypertrophy, the systolic murmur of mitral regurgitation is transmitted well into the left axillary region, and occasionally as far as the angle of the scapula. As compensation fails, the area of propagation of the murmur covers an ever-diminishing area of the chest wall. Moreover, in the case of the mitral systolic murmur the line of transmission does not follow the direction of the blood stream which calls the murmur into being, but is propagated toward the cardiac apex by the anterior papillary muscles and the wall of the left ventricle.

Time of Murmurs.—Every endocardial murmur bears a definite temporal relation to the events of the cardiac cycle, as they are generated during auricular systole, ventricular systole, or ventricular diastole. A murmur which is generated by ventricular systole is audible during this phase of the cardiac cycle and is termed a *systolic murmur;* while a murmur developing and audible during ventricular diastole is designated a *diastolic murmur.* Similarly, a murmur which is audible just prior to ventricular systole, or, in other words, during auricular systole, is termed a *presystolic murmur.*

Murmurs are further defined according to the portion of the individual phase of the cardiac cycle in which they are audible. Thus a *protosystolic* murmur is audible during the commencement of systole, a *meso-* or *midsystolic* murmur is audible during the middle of systole, while a *telesystolic* murmur is audible toward the end of the systole.

The time of endocardial murmurs is determined by inspecting or palpating of the cardiac impulse or the carotid artery while listening to the murmur, or by noting the temporal relation of the murmur to the first sound or to the long pause of the heart. The latter method is less accurate as it is difficult at times to determine the first sound, and the long pause is shortened in many cardiac diseases.

Quality of Murmurs.—Endocardial murmurs have been variously described as harsh, coarse and rasping, or as soft, blowing and musical. The diagnostic value of the quality of murmurs has been the subject of warm discussion for many years. In general, harsh and unmusical murmurs accompany stenotic lesions, while soft, musical or blowing murmurs characterize regurgitant lesions of the cardiac valves. But in the evaluation of the quality of individual murmurs it is to be borne in mind that the quality of a murmur is subject to change from time to time with changes in the size and structure of the orifice and valve segments, the caliber of the chamber of the heart into which the blood column is propelled, the degree of hypertrophy which is present, and the presence or absence of anemia. Moreover, when a murmur is generated in the heart which is adjacent to extensive pulmonary excavations containing air or to pneumothorax, or even in the presence of extreme tympanites or gastric distention, the murmur is apt to assume an altered quality of exocardial origin.

While studying the quality of the murmur, the examiner should also endeavor to determine whether the murmur is followed by the normal cardiac sound or whether it replaces this sound, as a murmur which merely accompanies or is superadded to the normal cardiac sound is not ordinarily of as grave prognostic significance as is a murmur which entirely replaces the sound of the heart.

Duration of Murmurs.—Murmurs may occupy any portion of the phase of the cardiac cycle in which they occur; they may be followed by a normal cardiac sound or may replace this sound; and their duration is measured by their relation to the duration of the several phases of the cardiac cycle.

The duration of a murmur frequently gives a clue to the degree of stenosis or regurgitation which is present. In the presence of adequate compensation, a brief "whiff" at a valve area, the murmur occupying only the earlier portion of the phase of the cardiac cycle in which it is audible, points to rapid expulsion through a relatively wide orifice; whereas, a prolongation of the murmur, usually with maintenance of the intensity or a crescendo effect, points to slow expulsion of blood through a narrow or stenotic orifice.

MITRAL MURMURS

Organic murmurs are generated at the mitral valve in the presence of stenosis and incompetence of the valve; and they are presystolic and systolic respectively, as they are audible just prior to or during ventricular systole.

Mitral Presystolic Murmur.—A presystolic murmur at the mitral area is indicative of mitral stenosis, the narrowing of the orifice whipping the blood stream into eddies which produce a murmur, which is audible just prior to the first sound of the heart. The murmur is commonly followed by a sharp, snapping first sound; but as mitral stenosis and regurgitation frequently coexist, the regurgitant murmur often masks or replaces the first sound of the heart at the apex.

The point of maximum intensity of the murmur is localized in the mitral area in the fifth left interspace over the cardiac apex, whence the murmur is not transmitted. The mitral presystolic murmur is loud, harsh and crescendo in quality, increasing in intensity to its abrupt termination, usually in a sharp first sound of the heart.

Mitral stenosis produces this purely presystolic murmur as long as left auricular hypertrophy is maintained. During the later stages of the disease a diastolic murmur is commonly audible, which has its inception immediately after the completion of the second sound of the heart. At the time of its appearance this murmur possesses a diminuendo character, occupying only the early portion of diastole, and early dying away. In certain cases, however, the diastolic murmur is merged into a concomitant presystolic murmur, with the consequent production of a continuous murmur, which persists throughout the diastolic period. This composite murmur is quite intense at its inception; it then diminishes in intensity to a variable degree; and it finally terminates in an ingravescent or crescendo presystolic phase. With failing compensation on the part of the left auricle, the presystolic element of the murmur is lost, but the diastolic portion remains audible, consuming a portion or the whole of the diastolic phase.

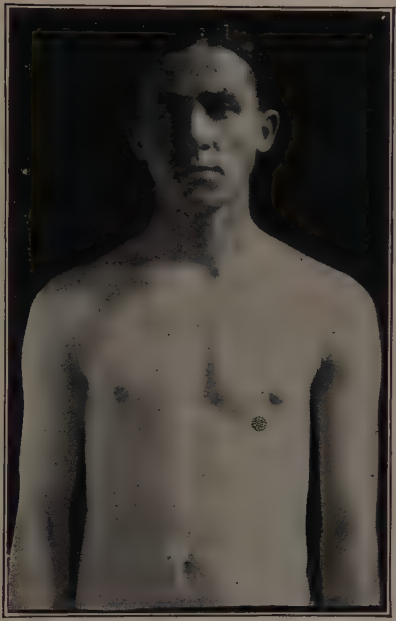


Fig. 227-*A*.—Point of maximum intensity of mitral presystolic murmur.

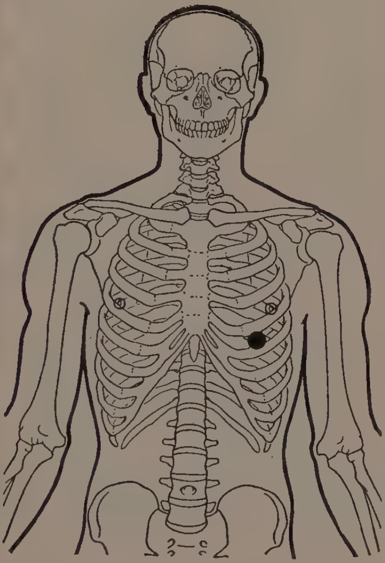


Fig. 227-*B*.—Point of maximum intensity of mitral presystolic murmur.

With the inception of auricular fibrillation the murmur of mitral stenosis is abolished.

The murmur of mitral stenosis is frequently accompanied by a palpable thrill over the cardiac apex; and the pulmonary second sound is commonly accentuated as a result of increased tension in the pulmonary circulation.

The murmur of mitral stenosis must be differentiated from the *Flint murmur,* which is also audible at the mitral area just prior to ventricular systole in cases of aortic regurgitation. The manner of generation of this murmur has not been adequately explained; but the accepted explanation is that in this disease the anterior cusp of the mitral valve becomes the target for two streams of blood entering the left ventricle from opposite directions, the one entering from the left auricle and the other regurgitating from the aorta, and is thereby thrown into vibrations, which are audible as a late diastolic or presystolic murmur.

The Flint murmur has its point of maximum intensity at the mitral area; it is audible during late diastole or just prior to systole; and it is not transmitted from the apical area. It has not the ingravescent or crescendo quality of the mitral stenotic murmur; it is not followed by a snapping first sound of the heart; it is rarely accompanied by a palpable thrill; and it has associated with it other signs of aortic regurgitation, as pulsation in the arteries of the neck, the water-hammer pulse, and occasionally the capillary pulse of Quincke.

In acute fibrinous pericarditis there is occasionally an audible presystolic friction sound which may be mistaken for the presystolic mitral murmur. Careful auscultation of the precordia will usually demonstrate that the point of maximum intensity of this sound is situated above the level of the cardiac apex.

The adventitious sound introduced into the cardiac cycle in presystolic gallop rhythm may likewise be confused with the presystolic mitral murmur.

Mitral Systolic Murmur.—Systolic murmurs generated at the mitral valve fall within two classifications; namely, *organic* murmurs due to deformity of the mitral valve from endocarditis or developmental defect, and *relative* murmurs, which are due to stretching of the mitral ring, whereby the borders of the cusps cannot be brought into close coaptation during ventricular systole. The two types of murmur present inherent characteristics which serve to differentiate the one from the other.

The point of maximum intensity of the organic mitral systolic murmur is localized in the mitral area over the apex of the heart, whence the murmur is transmitted toward the left axillary region, not infrequently as far as the angle of the scapula. In the case of this murmur the direction of transmission is not determined by the direction of the blood stream, which is regurgitating through the incompetent mitral orifice, but by the anterior papillary muscles and the wall of the left ventricle, which conduct the sound toward the apex of the heart.

The distance to which the murmur is transmitted is influenced by the size of the left ventricle and the transverse diameter of the thorax. Thus, the greater the hypertrophy of the left ventricle, and the more limited the transverse diameter of the thorax, the more nearly does the cardiac apex approach the lateral thoracic wall, and the farther toward the scapular angle is the murmur conducted.

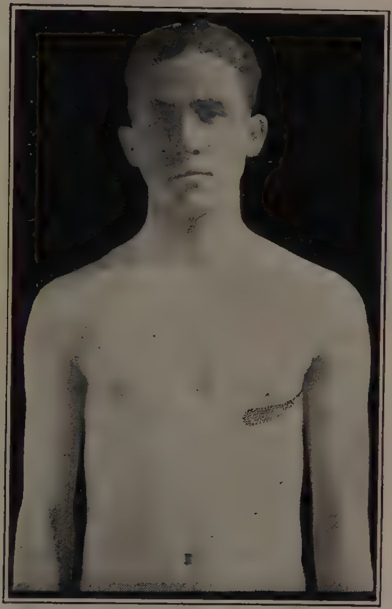


Fig. 228-*A*.—Point of maximum intensity and line of transmission of mitral systolic murmur.

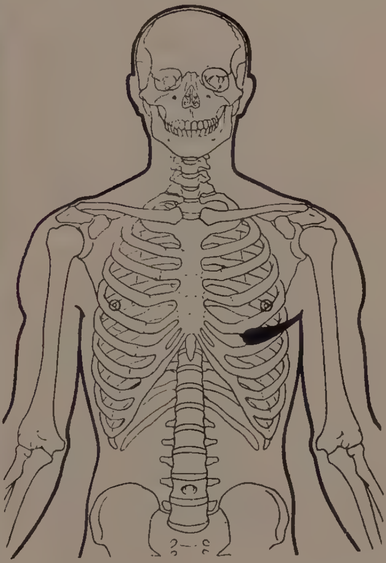


Fig. 228-*B*.—Point of maximum intensity and line of transmission of mitral systolic murmur.

The murmur occurs during ventricular systole. It may consume only the early or the latter portion of this phase of the cardiac cycle, or it may replace the first sound of the heart at the apex. In quality the murmur is commonly soft and blowing or musical and of low pitch. The intensity of the murmur varies with the state of the myocardium, remaining intense as long as compensation is maintained, and becoming less clearly audible or disappearing when dilatation is imminent or has occurred. The murmur is usually most clearly audible with the patient in the recumbent posture.

The murmur of mitral regurgitation is less constantly accompanied by a palpable thrill than is the murmur of stenosis of this valve.

The pulmonary second sound is accentuated in the majority of cases of organic mitral regurgitation, owing to right ventricular hypertrophy or occurring as a result of the heightened pressure in the pulmonary circulation; and, in long standing cases with marked changes in the mitral valve, a "safety-valve leak" frequently develops at the tricuspid valve.

The mitral systolic murmur of *relative mitral regurgitation,* occurring during the course of acute febrile diseases, anemic states, acute parenchymatous myocarditis, and after excessive physical effort, in which there is no structural deformity of the valve, but merely a stretching of the muscular ring at the base of the valve, is frequently evanescent in its manifestations, lacks the initial intensity of the organic mitral systolic murmur, and may not be attended by accentuation of the pulmonary second sound. It is not transmitted to any considerable distance from its point of maximum intensity.

AORTIC MURMURS

Murmurs generated at the aortic orifice are systolic and diastolic respectively, as they are occasioned by an obstruction to the free flow of blood from the ventricle into the proximal portion of the aorta during ventricular systole, or by lesions of the valve which by impairing its integrity permit a portion of the blood ejected during systole to regurgitate into the ventricle during diastole. Systolic murmurs occurring in the aortic area are likewise to be attributed in certain instances to aortic roughening and to dilatation of the proximal portion of the aorta, in the absence of stenosis of the aortic valve.

Aortic Systolic Murmur.—Stenosis of the aortic orifice is attended by a systolic murmur of striking intensity and harsh, unmusical quality, with its point of maximum intensity in the aortic area, in the second intercostal space adjacent to the right sternal border. The murmur is conducted from its site of production to its point of maximum intensity by the blood stream in the ascending aorta. In the presence of extreme grades of stenosis of the aortic orifice, associated with excessive left ventricular hypertrophy, the intensity of the murmur is such that it is audible over the entire precordia, and may readily be mistaken for a systolic murmur arising from the mitral valve. Careful auscultation of the precordia, however, will reveal the point of maximum intensity of the murmur in the aortic area.

From its point of maximum intensity the aortic systolic murmur is transmitted by the arterial stream into the carotid and subclavian arteries. In the absence of coincident incompetence of the aortic valve the stenotic murmur is followed by a slightly muffled second sound at the aortic area, the muffling of the sound in this instance contrasting in a striking manner with the purity of the second sound in the presence of a systolic murmur generated by changes in the proximal portion of the aorta. The aortic stenotic murmur is quite constantly attended by a palpable thrill.

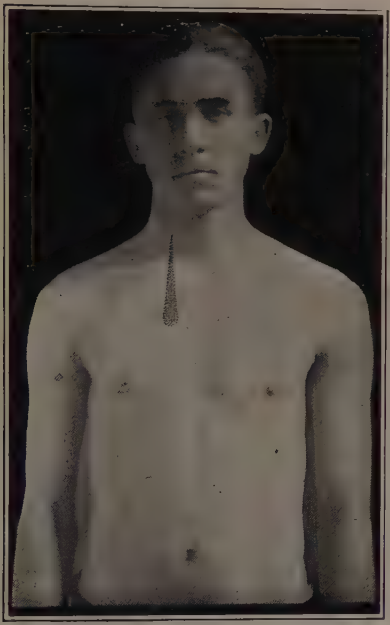


Fig. 229-*A*.—Point of maximum intensity and line of transmission of aortic systolic murmur.

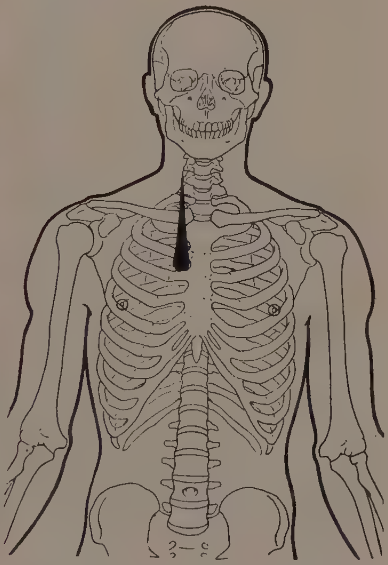


Fig. 229-*B*.—Point of maximum intensity and line of transmission of aortic systolic murmur.

The majority of systolic murmurs encountered at the aortic area are produced not by organic stenosis of the aortic valve, which is a comparatively rare lesion, but by roughening of the aortic cusps or by roughening or dilatation of the proximal portion of the aorta. The murmur arising from these causes does not possess the initial intensity of the aortic stenotic murmur; its quality is distinctly less harsh and unmusical than that of the stenotic murmur; and it is attended by a pure or accentuated second sound at the aortic area.

Aortic Diastolic Murmur.—A diastolic murmur with its point of maximum intensity at the aortic area is indicative of aortic regurgitation. The underlying lesion may be shrinking and thickening of the cusps, rupture or perforation of a cusp, or the presence of warty verrucosities upon the cusps, which prevent their accurate coaptation during ventricular diastole. Or again, the murmur may arise as the result of the inability of normal cusps to close an abnormally large aortic orifice, constituting in this instance *relative aortic regurgitation.* The aortic orifice, however, is usually surrounded by a fibrous ring so dense that stretching is of uncommon occurrence.

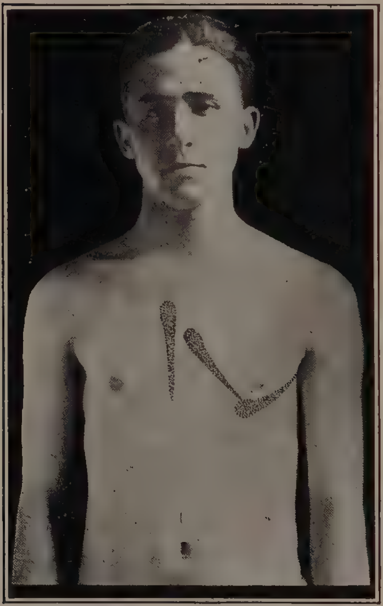


Fig. 230-*A*.—Points of maximum intensity and lines of transmission of aortic diastolic murmur.

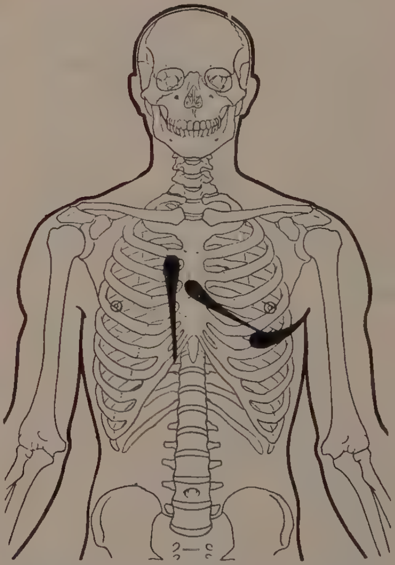


Fig. 230-*B*.—Points of maximum intensity and lines of transmission of aortic diastolic murmur.

The aortic diastolic murmur is most clearly audible at the aortic area, as a rule, and it is propagated thence in a direction ranging downward and obliquely across the sternum toward the cardiac apex. While the murmur is usually most intense at the aortic area, in certain cases it is to be heard most clearly over the upper portion of the gladiolus just below the Angle of Louis in the fourth intercostal space to the left of the sternum, or over the cardiac apex, whence it is transmitted downward and toward the left axillary region. The murmur occurs during diastole, masking or replacing the second sound of the heart at the aortic area. It is loud and blowing, but commonly is not harsh or unmusical as in the case of the aortic stenotic murmur.

While the murmur of aortic regurgitation may occur alone, it is not infrequently accompanied by a systolic murmur, due to coincident roughening of the valve or stenosis of the opening, a deformity of the aortic valve underlying both conditions. In such event a double murmur is generated at the aortic valve, which is harsh during systole, and less harsh or actually blowing and musical during diastole. Such a double murmur is apt to be confused during a casual examination with a pericardial friction sound with its to-and-fro rhythm, corresponding indefinitely with the systole and diastole of the ventricle.

TRICUSPID MURMURS

Murmurs are not frequently encountered at the tricuspid orifice; but when they are present, they are either presystolic or systolic in time, as they indicate stenosis or regurgitation at this orifice of the heart.

Tricuspid Presystolic Murmur.—The tricuspid stenotic murmur is audible in its maximum intensity in the tricuspid area over the lower portion of the gladiolus, whence it is not transmitted. It is due to tricuspid stenosis, which is usually of congenital origin. The murmur is commonly associated with a thrill over the tricuspid area together with an enfeebled pulmonic sound. The murmur is usually attended by moderate dyspnea, and by signs of general venous stasis.

Tricuspid Systolic Murmur.—A systolic murmur arising at the tricuspid valve is indicative of tricuspid regurgitation. The causative lesion may be a deformity of the cusps of the valve, arising as a sequence of ulcerative endocarditis; but more commonly a systolic murmur at this valve indicates *relative tricuspid regurgitation,* which is the result of increased blood pressure in the pulmonary circulation and engorgement of the right ventricle, occurring as the result of an obstructive disease of the lung or valvular lesions of the left heart.

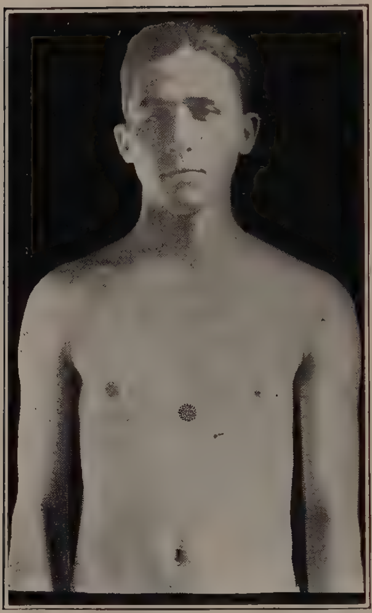


Fig. 231-*A*.—Point of maximum intensity of tricuspid presystolic murmur.

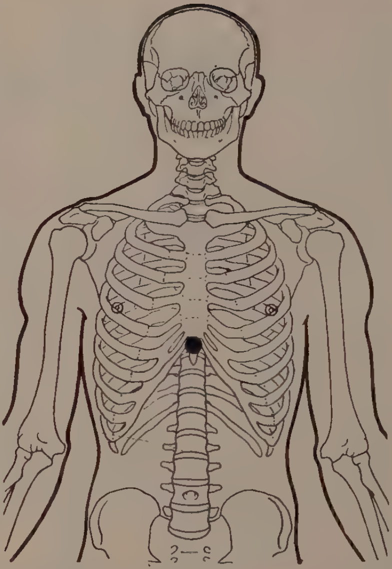


Fig. 231-*B*.—Point of maximum intensity of tricuspid presystolic mur mur.

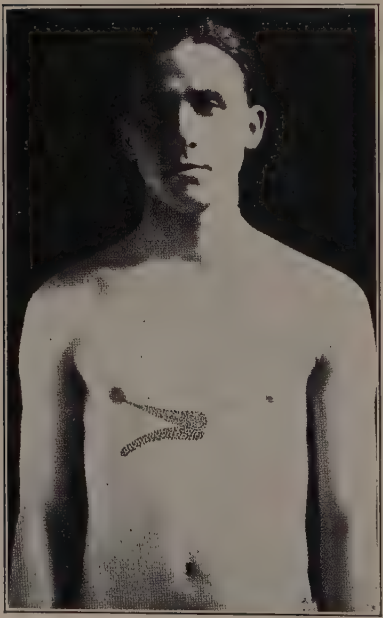


Fig. 232-*A*.—Point of maximum intensity and lines of transmission of tricuspid systolic murmur.

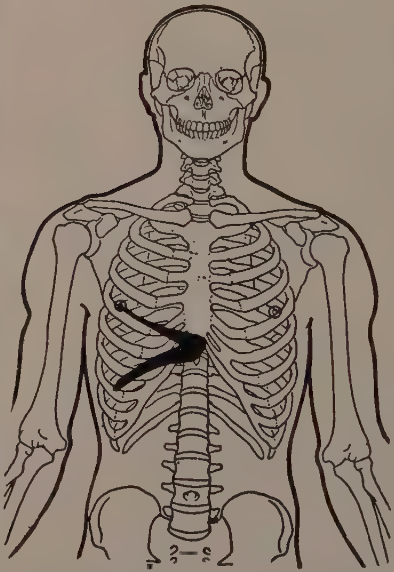


Fig. 232-*B*.—Point of maximum intensity and lines of transmission of tricuspid systolic murmur.

The point of maximum intensity of the tricuspid systolic murmur is not as definitely circumscribed to the valve area as is the case with murmurs arising at other valves of the heart; but careful auscultation of the precordia will usually serve to localize it in the tricuspid area, whence it is transmitted by the regurgitating blood stream toward the right and upward. Not infrequently the dilated or hypertrophied right ventricle lies in close apposition to the liver, in which event the murmur is transmitted downward and to the right as well as upward and to the right.

The first sound of the heart is obscured or replaced by the murmur in the tricuspid area. The pulmonary second sound varies in intensity with the concomitant state of the myocardium. In the presence of simple, isolated tricuspid regurgitation the second sound is unaltered in intensity or is slightly enfeebled, whereas in relative tricuspid regurgitation the second sound is accentuated during the maintenance of right ventricular hypertrophy, to become enfeebled with the supervention of right ventricular dilatation.

When relative tricuspid regurgitation develops during the course of mitral regurgitation, it is instructive to compare the relative intensity of the systolic murmurs occurring at the mitral and at the tricuspid valves. With failing compensation it will be noted that there is a progressive augmentation of the intensity of the tricuspid systolic murmur over that of the mitral murmur until the latter is masked entirely by the unduly intense tricuspid murmur.

The tricuspid systolic murmur is commonly blowing or whiffing in quality. In the presence of regurgitation of considerable magnitude there is to be noted systolic pulsation in the jugular veins and occasionally systolic pulsation of the hepatic region.

PULMONARY MURMURS

Murmurs at the pulmonary area are of very frequent occurrence, but organic disease of this valve is quite rare. The majority of systolic murmurs encountered at the pulmonary valve are functional, and will be discussed in a subsequent paragraph. Murmurs generated at the pulmonary valve are systolic and diastolic in time, as they are occasioned by stenosis or regurgitation at the valve.

Pulmonary Systolic Murmur.—An organic murmur at the pulmonary valve is occasionally encountered as an evidence of organic change in the valve. It is usually a sign of pulmonary stenosis from a congenital defect. The murmur occurs during ventricular systole; its point of maximum intensity corresponds accurately with the pulmonary valve area; it is harsh and unmusical when due to organic stenosis of the valve; and it is transmitted upward toward the root of the neck. The second sound of the heart as heard at the pulmonary area is impure or is replaced by a diastolic murmur.

The systolic murmur of *relative pulmonary stenosis,* which is audible in the presence of dilatation of the proximal portion of the pulmonary artery, is less harsh than is the pulmonary stenotic murmur, and is usually unattended by dyspnea, cyanosis or signs of right heart failure. In this case the pulmonary second sound is unimpaired or shows slight accentuation.

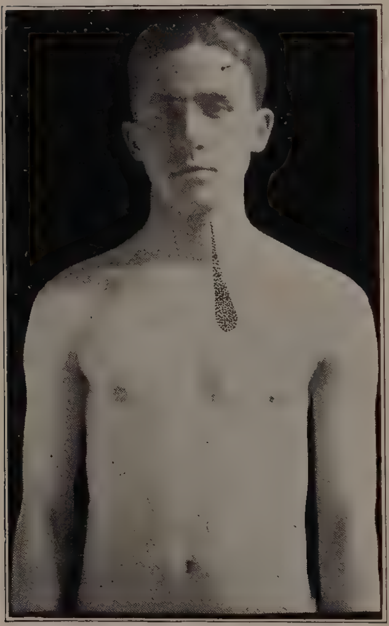


Fig. 233-*A*.—Point of maximum intensity and line of transmission of pulmonary systolic murmur.

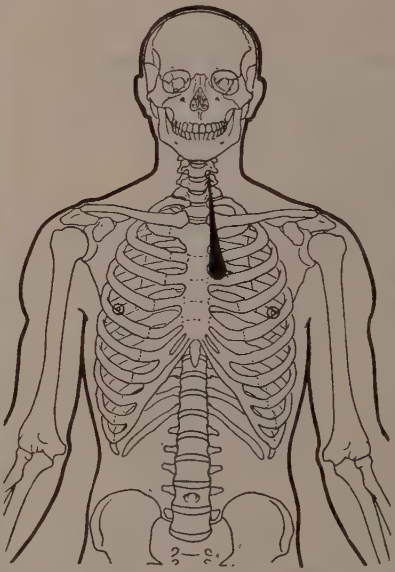


Fig. 233-*B*.—Point of maximum intensity and line of transmission of pulmonary systolic murmur.

The murmur of pulmonary stenosis may be confused during a casual examination with the systolic and diastolic bruit produced by patency of the interventricular septum, *Rogers' murmur.* This congenital defect of the heart is attended by a loud and rasping murmur with its point of maximum intensity in the fourth left intercostal space between the left sternal and midclavicular lines. The murmur is commonly attended by a palpable thrill. The murmur continues throughout systole and diastole, attaining its maximum intensity at the height of systole, and exhibiting a progressive diminution of intensity during ventricular diastole.

A systolic murmur with maximum intensity in the second or third interspace to the left of the sternum may be due to a patent ductus arteriosus. This murmur is not transmitted and is not accompanied by any change in the pulmonary sound. It may be attended by a systolic thrill.

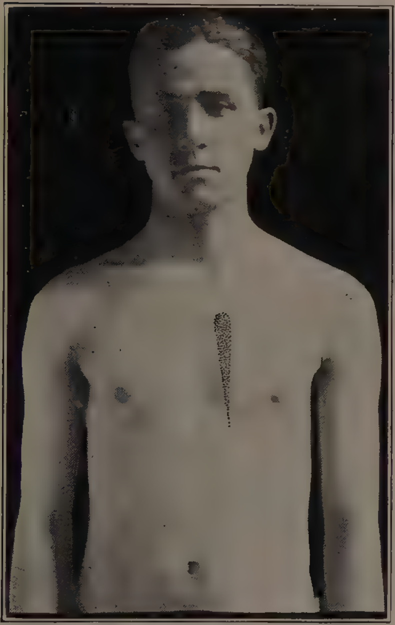


Fig. 234-*A*.—Point of maximum intensity and line of transmission of pulmonary diastolic murmur.

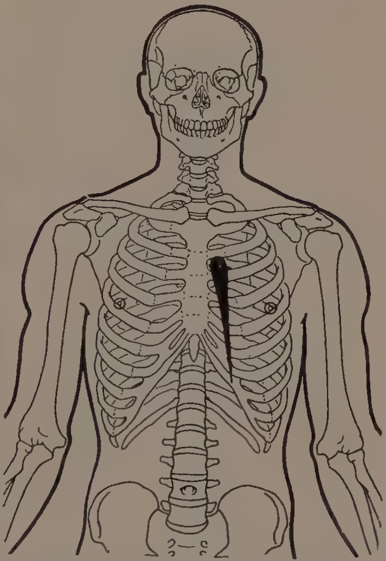


Fig. 234-*B*.—Point of maximum intensity and line of transmission of pulmonary diastolic murmur.

In the presence of fibroid retraction of the anterior border of the left lung, when an extensive area of the right ventricle is exposed directly to the anterior thoracic wall, the conus arteriosus of the right ventricle is likely to suffer compression during ventricular systole, with the consequent production of a systolic rumble in the conus. The murmur arising from the conus is obliterated during forcible inspiration, which causes the anterior pulmonary borders to intervene between the ventricle and the chest wall; and, moreover, the murmur is not attended by signs of embarrassment of the right heart.

Pulmonary Diastolic Murmur.—A diastolic murmur at the pulmonary area is significant of pulmonary regurgitation, due to alteration in the integrity of the cusps of the valve incident to ulcerative endocarditis, or occurring as a result of yielding of the muscular ring of the orifice so that the normal cusps do not close the abnormally large opening, *relative pulmonary insufficiency.*

The point of maximum intensity of the murmur is localized in the pulmonary area in the second left interspace, whence the murmur is propagated downward along the left sternal border. The murmur is audible during ventricular diastole, masking or replacing the second sound at the pulmonary area. In quality the murmur resembles the murmur of aortic regurgitation, save that it lacks the initial intensity of this murmur.

The *Graham Steell murmur* is a diastolic pulmonary murmur, audible in its maximum intensity in the third left interspace adjacent to the sternum, soft and blowing in quality, replacing the second sound of the heart in this area, and frequently audible only in the recumbent posture. It is significant of stretching of the muscular ring of the pulmonary valve and engorgement of the right ventricle. The murmur is frequently audible in late cases of mitral stenosis.

FUNCTIONAL MURMURS

Functional murmurs, which are also termed *inorganic, accidental,* and *hemic* murmurs, are endocardial bruits which arise in a heart which is not the seat of permanent structural change. Functional murmurs are not caused by valvular deformity; but they are attributed to excessive fluidity of the blood incident to anemia, to temporary myocardial weakness during the course of acute fevers, or to stretching of the valvular orifices as a result of excessive physical effort.

Functional murmurs are audible most frequently at the pulmonary area, and least frequently at the aortic area. They are more commonly encountered at the mitral than at the tricuspid area.

Functional murmurs are transient, present and absent at consecutive examinations, and do not persist for any considerable length of time. They occur during ventricular systole, but frequently do not consume the entire systole of the ventricle. Functional murmurs are soft, blowing and of low pitch, and are not transmitted beyond the limits of the precordia. When due to anemia they are often accompanied by arterial murmurs or the humming-top murmur in the jugular veins. Moreover, functional murmurs are not attended by changes in the character of the radial pulse, and are not accompanied by appreciable changes in the outline of the heart.

MULTIPLE MURMURS

While organic and functional murmurs arising at the various valves of the heart are separate entities and have been described singly, it is to be borne in mind that two or more murmurs may coexist in the same subject, the differentiation of which is not infrequently attended with considerable difficulty.

When two distinct murmurs are encountered at two valve areas, as for instance at the aortic and the mitral areas, the mere presence of two murmurs at two valves does not necessarily signify organic disease of both valves, as one murmur may be relative. Thus, organic regurgitation at the aortic valve may be attended by dilatation of the left ventricle and yielding of the mitral ring. The mitral murmur in this instance is due to relative mitral regurgitation. Similarly, in organic mitral insufficiency there is not infrequently an associated relative tricuspid regurgitation.

When two murmurs occur at two phases of the cardiac cycle, one systolic and the other diastolic, this fact is of considerable aid in the differential diagnosis. When, however, two murmurs occurring at the same phase of the cardiac cycle are discovered, the differentiation must rest largely upon the points of maximum intensity and the lines of transmission of the murmurs. The quality of the murmurs in this instance is of some assistance, remembering the general rule that stenotic murmurs are harsh and unmusical, while regurgitant murmurs are generally soft, blowing or musical. If, on the contrary, both murmurs are alike in quality, it is probable that there is only one murmur, which is transmitted from the orifice where it is generated to a second valve area, as in the case of the transmission of the murmur of aortic regurgitation to the apical area of the heart.

Moreover, murmurs arising in the heart must be differentiated from a possible cardiorespiratory murmur by directing the patient to suspend respiration, whereupon the latter will disappear. The differential points between endocardial murmurs and the pericardial friction sound are detailed in a subsequent paragraph Finally, in the differentiation of multiple murmurs the auscultatory findings must be correlated with the general appearance of the patient and such accessory signs as edema, dyspnea and cyanosis.

THE CARDIORESPIRATORY MURMUR

When a segment of the anterior border of the lung is anchored by pleural or pleuropericardial adhesions between the ventricle and the thoracic wall, and when during the course of hypertrophic emphysema, chronic ulcerative phthisis, or massive pleural effusion the anterior borders of the lungs intervene between the heart and chest wall, there is frequently generated a systolic "blow" or "whiff," which is detected near the cardiac apex, along the left border of the heart or even to the right of the sternum, the *cardiorespiratory murmur*. The sound elicited under these circumstances should in all cases be assigned to its real cause; namely, the sudden expulsion of air from a portion of the lung by the impact of the heart during systole. Occasionally the sound is encountered in the absence of pulmonary disease when the left ventricle is excessively hypertrophied.

The intensity of the cardiorespiratory murmur is generally accentuated upon deep inspiration; and the murmur not infrequently becomes inaudible upon forced expiration, as a result of the expiratory recession of the anterior pulmonary borders. Not infrequently the murmur is suppressed when the subject assumes the recumbent posture. While the adventitious sound occurs during ventricular systole, it does not coincide accurately with the commencement or termination of this phase of the cardiac cycle, but is usually audible during the middle of systole.

PERICARDIAL FRICTION

In the presence of inflammation of the pericardium the surfaces of the visceral and parietal membranes, which glide noiselessly over each other during health, become roughened, and a friction sound is generated, the intensity and quality of which depend upon the degree of pericardial inflammation and the power of the cardiac contractions. Depending upon the state of the pericardial surfaces, a sound is produced which varies in quality from that of the sound which is produced upon gently stroking a silken fabric to a loud, rasping sound similar to the sound produced by the creaking of new leather. The pericardial friction sound corresponds roughly with the sounds of the heart, but not with the same accuracy as do endocardial murmurs, as the phases of the friction sound last longer than do the normal heart sounds. To these sounds there may be added a presystolic friction sound occurring during auricular systole.

As a rule, the friction sound is most distinctly audible along the left sternal border between the second and fourth intercostal spaces, though in certain instances it is limited to the region of the cardiac apex.

In differentiating pericardial friction from endocardial murmurs, the examiner should bear in mind the inherent differences between the two phenomena. Endocardial murmurs bear a far more definite relation to the events of the cardiac cycle than do the phases of the pericardial friction sound. Pressure exerted upon the precordia by the bell of the stethoscope frequently serves to accentuate the intensity of pericardial friction, but the same maneuver is without effect upon the intensity of endocardial murmurs.

Forced inspiration exerts quite a different influence upon the sounds emanating from the two sources. Thus, full inspiration by exerting pressure upon the pericardial surfaces intensifies the pericardial friction sound, while the same act causes a diminution in the intensity of endocardial murmurs by the introduction of the anterior borders of the lungs between the heart and the thoracic wall. While many endocardial murmurs are transmitted far beyond the limits of the precordia, pericardial friction is limited to the precordia, and frequently it is audible over only a restricted portion of this area.

Changes of posture exert a more striking influence upon the intensity of pericardial friction than is the case with endocardial murmurs. The friction sound is intensified by bending the trunk forward, and the sound frequently disappears when the subject assumes the recumbent posture. Upon auscultation of the precordia, the impression is conveyed to the examiner that the pericardial friction sound is generated immediately beneath the integument, whereas endocardial murmurs seem rather to come from the depths of the thorax. Moreover, pericardial friction is an evanescent sound, frequently present and absent upon consecutive examinations, and changing in intensity from hour to hour, changes which are not observed in organic murmurs of the heart.

In certain cases the outline of the area of cardiac dullness is of assistance in the differentiation, that of pericarditis differing essentially from that which attends the cardiac enlargement incident to chronic valvular disease.

Pericardial friction must be differentiated from certain substernal sounds of doubtful etiology. In the presence of cardioptosis the cardiac sounds, as elicited over the lower sternal region, frequently possess a harsh scraping or scratching quality, the *xyphosternal crunch*, which may be mistaken for the pericardial friction sound. Of uncertain etiology, this sound is detected in a good many normal subjects.

Pleuropericardial friction, generated in the presence of inflammation of the pleura in relation with the pericardium, is usually to be detected along the left border of the precordia or in the region of the cardiac apex. The sound in this instance bears a definite relation to the movements of the heart; but it is also influenced by the excursions of the anterior pulmonary borders. If the area of inflammation is situated upon the visceral pleura of the left lung, in contact with the pericardium, the sound is abolished during forced expiration, at which time the lappet of lung recedes from the heart. Again, if the area of inflammation is situated upon the costal pleura, anterior to the heart, during forced inspiration the sound is enfeebled or abolished by the intervention of the anterior border of the left lung between the pericardium and the inflamed pleura.

PERICARDIAL SUCCUSSION SOUND

In the presence of air and fluid in the pericardium during the course of hydropneumopericardium, a medley of splashing and gurgling sounds is generated by the movements of the heart, the *pericardial succussion sound.* The sounds in certain cases possess a metallic consonance, hence the name *metallic gurgle,* sometimes employed to describe them. In other instances the sound is analogous to that which is produced by a water-wheel in motion. The sound is occasionally audible without the aid of the stethoscope; and, upon auscultation of the precordia, its intensity may completely obscure the cardiac sounds. The sound is not influenced by the suspension of respiration.

VASCULAR MURMURS

As endocardial murmurs are generated by deformities of the valves of the heart and by alterations in the density of the blood, so also under somewhat similar conditions murmurs are generated in the arteries and veins.

ARTERIAL MURMURS

Auscultation of the arteries may reveal the presence of murmurs in the aorta, the carotid, subclavian, brachial and femoral arteries. During auscultation of peripheral arteries the stethoscope should be applied evenly to the integument but not with sufficient force to diminish the lumen of the vessel, as constriction of the lumen will engender an audible bruit in the normal artery.

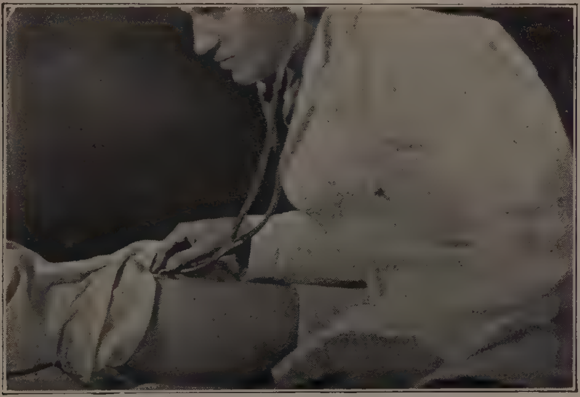


Fig. 235.—Auscultation of femoral artery.

The Aorta.—Upon auscultation of the aorta in the left interscapular region a systolic murmur in the vessel is a sign of aneurysm of the aorta. The murmur is accompanied by the concomitant signs of aneurysm; namely, dullness on percussion, frequently a palpable thrill, pulsation of the chest wall in many instances, and diverse pressure symptoms. Frequently there is a palpable tracheal tug. The x-ray reveals an area of density over the portion of the aorta affected. Fluoroscopic examination often reveals pulsation of the dense area.

The Carotids.—Upon auscultation of the carotid artery the first and second sounds of the heart may sometimes be heard, although the first sound is more frequently inaudible. These sounds are not to be confounded with murmurs, as they are merely the normal sounds of the heart which are transmitted along the course of the blood stream. Endocardial murmurs, however, are similarly transmitted, a harsh systolic murmur audible over the vessel signifying aortic stenosis, aortic roughening or aneurysm of the arch. The transmitted second sound of the heart may be replaced by the diastolic murmur of aortic regurgitation.

The Subclavian Artery.—In certain cases of apical pulmonary tuberculosis a systolic murmur is audible over the course of the subclavian artery. The murmur in this case is due to constriction or to bending of the lumen of the vessel by the traction of pleural adhesions. A diastolic murmur is occasionally audible in the subclavian artery in aortic regurgitation.

The Femoral Artery.—In many cases of aortic regurgitation a double murmur, systolic and diastolic, is to be elicited by auscultation of the femoral artery. The systolic murmur results from the sudden injection of blood into the aorta; while the diastolic murmur results from the reflux of blood during diastole, since at this time the intraventricular pressure is lower than the pressure in the peripheral circulation. This double murmur constitutes *Duroziez's sign* of aortic regurgitation. Pressure with the bell of the stethoscope sufficient to slightly indent the artery will produce a systolic murmur in the normal subject, but if there is sufficient back flow in diastole the diastolic murmur of Duroziez will be accentuated. Friedreich elicited the sign with great constancy in aortic regurgitation, but also found it present with aneurysm of the aorta and in left ventricular hypertrophy with competent aortic valves.

VENOUS MURMURS

A continuous murmur may be elicited over the jugular vein in the normal subject by tightly applying the bell of the stethoscope to the integument over the course of the vessel. A similar murmur may occasionally be elicited by turning the head far to one side, due to constriction of the jugular vein by bands of the cervical fascia and the omohyoid muscle. Hence, during auscultation of this vessel the stethoscope should be lightly applied to the integument, and the head should be maintained in a symmetrical position.

The Venous Hum.—The principal diagnostic sign afforded by auscultation of the venous system is the *venous hum, humming-*

top murmur, or *nun's murmur*. This murmur which is continuous, has been compared to the sound of the buzzing of insects and to the sound of a circular saw in action, comparisons which give but an imperfect conception of the quality of the sound.

The sound is commonly to be elicited over both jugulars, but it is more intense, as a rule, over the right vein. The murmur has its maximum intensity over the inner third of the clavicle. The intensity is increased when the patient assumes the upright posture, during inspiration, and during diastole, factors which favor a more rapid flow of blood through the veins.

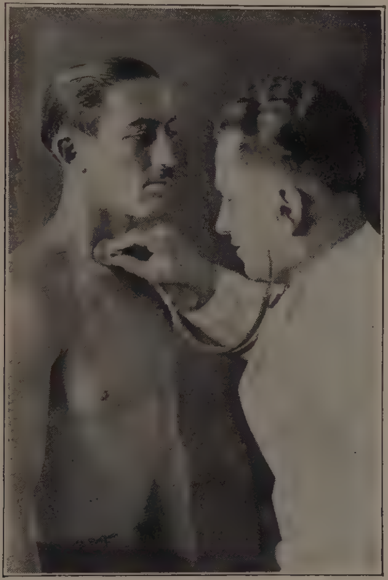


Fig. 236.—Auscultation of jugular vein.

The murmur is generated in the bulb of the jugular vein and not in the carotid artery, as described by Laënnec. The intravenous origin of the murmur is demonstrated by the facts that the murmur is continuous and is not intermittent as are arterial bruits, and that it can be entirely suppressed by light compression of the jugular vein, a compression too insignificant to appreciably influence the action of the carotid circulation.

The venous hum is occasionally to be elicited in the young subject during health; but in the vast majority of cases its presence points to anemia, especially chlorosis and pernicious anemia.

Friedreich has detected a similar venous murmur in the right interscapular region at the level of the third and fourth dorsal vertebræ, which occurred in conjunction with the venous hum in the jugular veins, and which he attributed to excessive fluidity of the venous content of the superior vena cava. Murmurs of tricuspid regurgitation are sometimes transmitted into the veins of the neck.

CHAPTER XXIX

AUSCULTATION OF THE ABDOMEN

Auscultation is seldom employed in the examination of the abdomen and its viscera. Upon auscultation of the abdominal surface overlying the liver or spleen, a friction sound may occasionally be audible in the presence of perihepatitis, subdiaphragmatic abscess, or of perisplenitis. Similarly, vascular murmurs have occasionally been encountered upon auscultation of the abdomen overlying these organs, as a result of transient constriction of their extensive vascular channels. In tricuspid regurgitation the murmur is often well transmitted to the liver. In cases of aneurysm of the abdominal aorta a vascular murmur may be detected by auscultation over the course of this vessel. During pregnancy, auscultation of the fetal heart sounds and uterine souffle should be practiced at frequent intervals.

THE STOMACH

Succussion sounds which are generated in the stomach and which are audible during palpation possess a correspondingly greater intensity upon auscultation of the epigastrium, and are propagated to a considerable distance from their site of production. In eliciting these sounds by auscultatory percussion, the patient should assume the dorsal decubitus with the abdominal muscles relaxed. The bell of the stethoscope is placed over the central portion of Traube's space and the epigastrium is gently tapped with the finger tips of the opposite hand of the examiner, whereupon the examining ear appreciates a series of sounds analogous to those which are produced upon shaking a rubber bag which is partially filled with fluid. Demonstrable in the normal stomach when it contains coincidentally air and fluid, the succussion sound possesses a much greater range of intensity and area of propagation in the case of gastrectasis.

Upon auscultation of the stomach in the presence of gastrectasis with active fermentation of the gastric contents, the examiner frequently encounters a series of fine crackling sounds, caused by the bursting of minute bubbles upon the surface of the contained fluid, the enlarged stomach amplifying and acting as a resonator for the sounds. These sounds are never audible below the inferior curvature of the stomach; and, when they are demonstrable in the presence of a dilated stomach, serve as a reliable guide in checking the findings upon palpation and percussion of the organ.

Kronicker and Meltzer first called attention to two deglutition sounds which are audible upon auscultation of the superior portion of the epigastrium immediately below the tip of the ensiform cartilage. The first of these sounds is simultaneous with the act of deglutition, while the second which is attributed to the passage of the ingested fluid through the cardiac orifice, is audible approximately seven seconds after the first sound. In the presence of partial stenosis of the cardiac orifice of the stomach the second sound is replaced by an irregular spouting, churning sound as the ingested fluid passes through the constricted orifice. In the presence of complete stenosis of the cardia, the second deglutition sound is abolished.

THE SMALL INTESTINE

Upon auscultation of the abdominal wall over the distribution of the small intestine, the examiner frequently encounters transient gurgling sounds, *borborygmi,* which are without diagnostic significance. But in the presence of a partial obstruction of the intestine, auscultation at the level of the stenosis reveals a more intense fluid sound, resembling that which is produced by the sudden expulsion of liquid from a syringe. In the presence of multiple partial intestinal stenoses due to tuberculous ulceration König has encountered these fluid sounds at varying levels of the abdominal wall, accompanied by succussion sounds upon palpatory percussion and by the sloping dullness of Mathieu and Ricard.

THE LARGE INTESTINE

The chief value of auscultation in connection with the examination of the large intestine is in auscultatory percussion in differentiating the gastric and the colonic tympany. Auscultation frequently reveals the presence of gurgling sounds over the cecum in the presence of typhoid fever, which are in no wise pathognomonic of this disease. Similarly, in the presence of severe diarrhea or dysentery gurgling is encountered over the sigmoid flexure.

Very rarely is the examiner able to detect upon auscultation a peritoneal friction sound in the presence of inflammation of the walls of the colon or in the presence of miliary tuberculosis or malignant disease of this tract.

SECTION VII

SENSORY PHENOMENA AND REFLEXES

CHAPTER XXX

SENSORY PHENOMENA

An accurate sensory examination requires the free and full cooperation of the subject of the examination and a refined technic upon the part of the examiner. The area under examination should be freely exposed and the examination should be conducted in a chamber which is free from noise and in which the temperature is constant and is neither uncomfortably elevated nor lowered. The sensory examination should not be unduly prolonged; but when any disorder of sensation is detected, its exact limits should be determined. In this connection it is important that the examiner should refrain from graphically charting the limits of the area of dysesthesia upon the skin, as such practice serves as a fertile source of suggestion to the subject of the examination.

From the course of the afferent sensory pathway it is evident that a sensory disturbance may be due to a lesion involving the integrity of a peripheral nerve trunk, involving one or more of the tracts of the spinal cord, or involving the brain stem or the cerebral cortex. The area and distribution of the sensory disturbance will in each event serve to localize the site of the causative lesion. Disturbances of general sensation comprise disorders in the sense of touch, pressure sense, the senses of temperature and pain, and of muscular sense, which comprises the perception of active and passive movements.

Tactile Sensation.—The acuity of tactile sensibility is tested by gently touching the cutaneous surface in various regions with a feather, a camel's-hair brush, or with a twisted wick of cotton, while the subject's eyes are bandaged. Tactile sensation is also investigated by detecting the minimal distances at which the two points of a compass may be appreciated when simultaneously placed upon the skin of the subject.

The normal subject can state the precise instant at which the integument is touched and, in a general way, the nature of the fabric with which it is in contact. The readiness with which the two compass points are recognized varies widely in different regions of the body, being most sensitive upon the tip of the tongue, where they are recognized when separated by only one millimeter, and least readily detected upon the back, arm, and thigh, where they are only recognized when separated by a space of from 60 to 80 millimeters. While the readiness with which tactile sensations are registered is a reliable index to the integrity of the sensory pathway, allowance must be made in certain instances of delayed transmission for the degree of natural intelligence of the subject of the examination.

Moderate impairment of tactile sensibility constitutes hypoesthesia, indicating in the majority of cases a compression or partial lesion of the sensory pathway. An abnormally acute perception of tactile sensation constitutes hyperesthesia, which frequently indicates a functional irritability of the sensory pathway. A complete abolition of tactile sensibility over a zone of the body constitutes anesthesia. A circumscribed anesthesia is likely to prove of peripheral origin, whereas an extensive zone, embracing one-half of the body or the entire body below a definite level is of central origin.

In the presence of a complete transverse lesion of the spinal cord there is anesthesia and motor palsy which is bilateral below the level of the lesion. In the presence of a hemisection of the cord or of a lesion involving only one lateral half of the cord, there ensues the phenomenon which is designated Brown-Séquard paralysis, in which there is complete motor palsy upon the corresponding side below the level of the lesion, with complete anesthesia and partial loss of motor power upon the side opposite to the cord lesion.

Pressure Sense.—The pressure sense is investigated by noting the ability of the subject to appreciate minor variations in pressure, when cubes of uniform size but of varying weight are placed upon the surface under examination. During this examination muscular sensation is to be eliminated by placing the limb upon a firm, unyielding surface; and temperature sense must be excluded, as extremes of temperature have a tendency to impair the nicety of the pressure sense perception. Variations in pressure sense, which possess the same significance as do similar variations in tactile sensation, are not relied upon as much as are the latter in neurologic examinations.

Sense of Temperature.—The entire cutaneous surface is supplied with specific "heat spots" and "cold spots," which are supplied by nerve endings for the appreciation of these extremes of temperature sense. Hence the power of discriminating variations in temperature may be retained, while tactile sensation is temporarily or permanently abolished. Compression of the ulnar nerve, which causes a marked diminution of tactile sensibility over the distribution of the nerve, does not involve the temperature perception in this area. As a general rule, the portions of the body which are habitually clothed are more sensitive to thermic variations than are the exposed portions of the integument.

The cutaneous perception of temperature is most conveniently tested by the application to the area under examination alternately of test tubes containing water at temperatures considerably above and below the body temperature. In the presence of organic disease of the spinal cord, as syringomyelia and in the presence of lesions of the medulla and pons, as hemorrhage, tumor, or softening, the perception of temperature is impaired (thermohypoesthesia). Similarly destructive lesions in these regions result in total abolition of the perception of changes of temperature, constituting thermoanesthesia; whereas functional irritability of the tract of Gowers may result in an exaggerated perception of variations in temperature, constituting thermohyperesthesia.

Sense of Pain.—While it is true that any form of appreciable sensation when sufficiently magnified and intensified may assume such proportions as to render its perception painful, it is yet generally agreed that there are specialized "pain spots" distributed universally over the cutaneous surface, which are supplied by special nerve endings; and that the sensation of pain when pronounced is not invariably due to overstimulation of the fibers having to do with tactile or temperature sensation, but to hyperstimulation of these specific pain centers in the integument.

The perception of the sense of pain is investigated by pricking the integument of the area under examination with a sharpened quill or with an ordinary pin or needle. In the presence of central cord lesions which are incomplete, in posterior column sclerosis of the cord and in lesions of peripheral nerves, the perception of painful stimuli may be delayed and impaired, constituting hypalgesia, or may be totally abolished in the area under investigation, constituting analgesia. Similarly in the presence of functional nervous states the examiner is apt to encounter an exaggerated perception of painful stimuli, constituting in this event hyperalgesia.

Different forms of perverted sensation may be encountered, which are grouped under the term parasthesiæ. These perversions are principally subjective in character and are not modified or influenced by objective examination of the subject. They may assume the character of the crawling of insects (formication); they may take the form of alternate flashes of heat and of cold; or the subject may complain of transient numbness. Occasionally upon painful stimulation of one extremity there is experienced coincidentally a painful sensation in a symmetrical distribution of the opposite limb, constituting in this instance, allocheiria.

Muscular Sense.—Muscular sensation is the peculiar sense by means of which judgments are formed as to the weight of articles which are lifted, by which the patient is aware of the position of certain portions of the body without the aid of the eyes, and by which he is enabled to maintain the standing posture without conscious effort.

The muscular sense is investigated by noting the perception of active and passive movements of the limbs. Thus, the subject is directed to perform various movements with the limbs, such as describing a semicircle on the floor with the toe, or touching the knee with the ankle of the opposite limb. In testing the perception of passive movements, the limb of the patient is slowly moved, while the eyes are lightly bandaged, and he is asked to indicate by pointing the range of movement and the new position of the limb.

Stereognostic Sense.—Stereognostic sensibility is the faculty by which objects placed in the hand are recognized by their palpable shape and consistence. An abolition of this sense (astereognosis) is frequently indicative of a lesion involving the superior parietal lobule of the brain.

CHAPTER XXXI

THE REFLEXES

The Reflex Arc.—The simplest form of reflex arc comprises an afferent or sensory neurone, which conducts impulses from the periphery of the body to an intermediate cell station situated in the gray matter of the spinal cord, and an efferent motor neurone, over which the intermediate cell station or medullary center discharges impulses in response to the sensory stimulus which is conveyed to it by way of the afferent sensory neurone. The entire sequence of changes which ensues upon adequate stimulation of the receptor of the afferent sensory neurone constitutes the reflex act.

In the case of the spinal reflexes the afferent neurone is represented by the peripheral spinal sensory nerve with its root ganglion, the intermediate cell station by the cells of the gray matter of the spinal cord, and the efferent neurone by the motor nerve arising from the cells of the anterior horn of the cord. The shorter branch of the dorsal nerve root upon entering the posterior horn of the spinal cord gives off collaterals which pass immediately to form synapses with the cells of the anterior horns of the same side of the spinal cord, a reaction in this instance constituting a homolateral spinal reflex. However, the spinal nerve root also furnishes collateral branches which pass by way of the anterior white commissure to form synapses with the anterior cornual cells of the opposite side of the cord, resulting in contralateral reflexes. Also certain collateral branches form synapses with motor cells in higher and lower levels of the spinal cord. A much more complicated reflex is represented by the collaterals which establish connections with the cells of origin of the columns of Goll and Burdach, by which they are relayed to the gracile and cuneate nuclei of the medulla, thence by the arcuate fibers to the opposite optic thalamus, and thence by the third sensory neurone to the sensory area of the cerebral cortex. In this situation an intermediate cell station in the form of association bundles is established, the efferent motor neurone of this extensive reflex arc being represented by the pyramidal tracts, the reflex action resulting in consciousness and in volitional muscular movement. Another very complex reflex arc is formed by the dorsal root fibers which form synapses with the cells of Clarke's column, whence the impulse is relayed by the ascending spinocerebellar tract of Flechsig to the cerebellar cortex and dentate nucleus, thence to the red nucleus of the midbrain, and thence by the rubrospinal tract to the anterior cornual cells, the reflex act in this instance having for its expression the regulation of muscle tone and the synergic action of different muscles in the maintenance of equilibrium.

Simple spinal reflexes occur without any intervention upon the part of the cerebrum, the various spinal segments acting independently; but there are governing fibers descending in the pyramidal tracts from the cerebral cortex, which modify and may volitionally inhibit the spinal reflexes.

Reflexes are variously classified as tonic and clonic, as superficial reflexes and as deep reflexes. The principal superficial reflexes are the plantar, cremasteric, and abdominal reflexes; while the principal deep reflexes comprise the patellar and achilles tendon reflexes, the jaw-jerk, the ankle clonus and the patellar clonus.

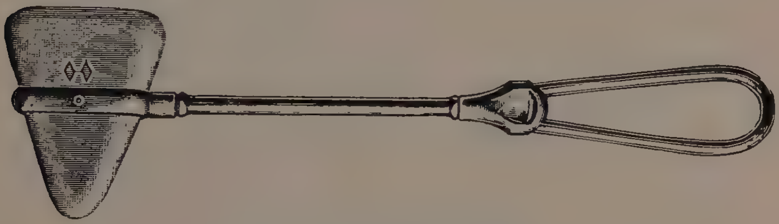


Fig. 237.—Reflex hammer.

Abdominal Reflex.—The abdominal reflex consists in a contraction of the underlying abdominal muscles when the integument is stimulated, as for instance, by gently scratching with a sharp instrument. This reflex should be elicited upon either side of the abdomen both above and below the level of the umbilicus, in order to demonstrate the independence of the upper portion of the rectus, which is supplied by the eighth and ninth intercostal nerves; and the lower portion of the rectus, which is supplied by the tenth and eleventh intercostal nerves. In each case, the umbilicus is observed to be drawn toward the side of the stimulation in the presence of an intact reflex. It may readily be demonstrated that in the case of the normal subject the umbilicus is not displaced upon changing from the dorsal recumbent to the sitting posture. But in the presence of paralysis of the lower portion of the rectus, upon the change of posture the umbilicus is elevated, thus differentiating the lesion of the tenth and eleventh intercostals from the integrity of the eighth and ninth intercostal nerves above the umbilicus.

Cremasteric Reflex.—Upon stimulation of the integument upon the inner side of the thigh the testicle of the corresponding side is elevated. The segments which preside over the cremasteric reflex comprise the first and second lumbar segments.

Plantar Reflex.—In the normal subject, when the sole of the foot is stroked with a probe or a sharp instrument, plantar flexion of all of the toes ensues as the reflex response. In the presence of a lesion or disturbance of function of the upper motor neurone, instead of the uniform plantar flexion of the toes, the great toe is extended while the remaining toes are flexed or separated from each other (Babinski's sign). In eliciting the plantar reflex the outer portion of the sole should be stroked in a direction from before, backward. By stroking the skin below the external malleolus a similar reflex is obtained (Chaddock's sign). This reflex is usually but not always present with the Babinski reflex and is at times obtained when the Babinski sign is absent. Subjects yielding Babinski's and Chaddock's signs sometimes also yield, upon proper stimulation, the reflexes of Oppenheim and of Gordon.

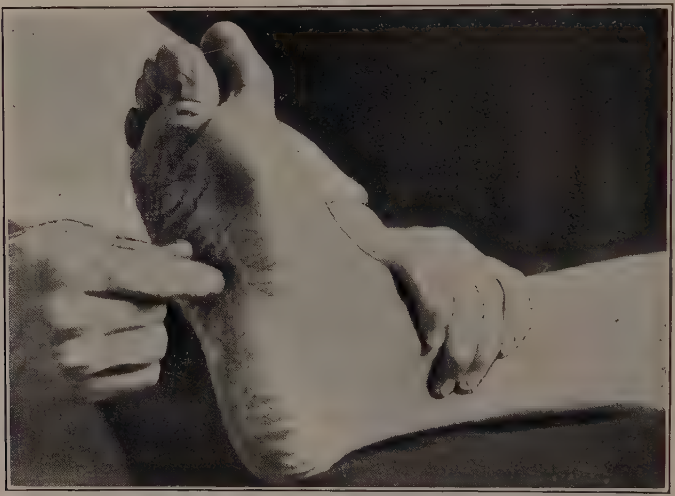


Fig. 238.—Elicitation of Babinski's sign.

Oppenheim's Reflex.—Oppenheim's reflex is elicited by passing the thumb or the handle of the percussion hammer downward along the internal border of the tibia, at the same time exerting pressure upon the soft tissues, whereupon extension of the great toe with plantar flexion or separating of the remaining digits ensues. The significance of Oppenheim's reflex is identical with that of the pathologic plantar reflex.

Gordon's Reflex.—If, instead of proceeding as in the case of Oppenheim's reflex, the examiner merely grasps the muscles of the calf of the leg and exerts deep pressure, in the presence of a lesion of the upper motor neurone, extension of the great toe ensues.

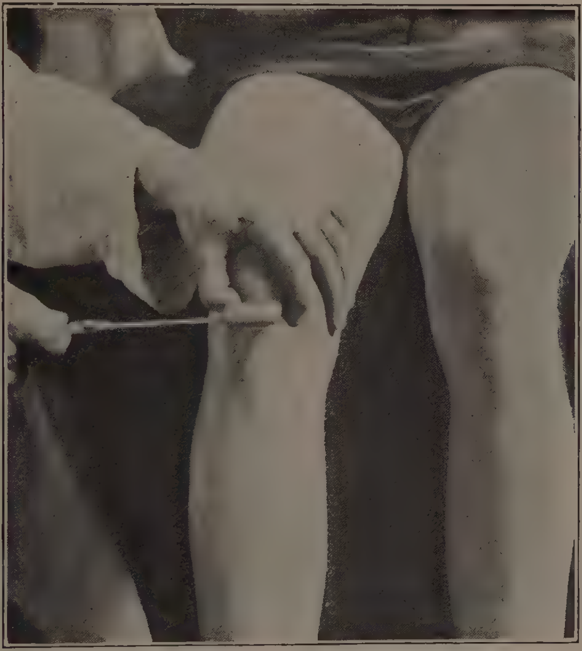


Fig. 239.—Elicitation of patellar tendon reflex.

The Patellar Tendon Reflex (Knee-jerk).—If the patient is in the sitting posture, to elicit the knee-jerk the leg is flexed upon the thigh at a right angle, while the patellar tendon is struck a rapid light blow with the reflex hammer. If the patient is bedridden, the reflex may be elicited by raising the leg from the bed by means of a hand placed beneath the knee joint while the blow is delivered.

During the examination every effort should be made to distract the attention of the patient from the procedure in order to prevent cerebral inhibition of a normal reflex. If the subject is very selfconscious and the reflex consequently is elicited with difficulty, Jendrassik's reinforcement may be resorted to. The patient is directed to lock the hands and to pull, meanwhile keeping his eyes fixed upon the ceiling. The reflex arc of the patellar tendon reflex comprises the second, third, and fourth lumbar segments.

Exaggeration of the patellar tendon reflex may be indicative of disease between the level of the reflex arc and the cerebral cortex, whereby the governing impulses from the cerebrum are interrupted. Such a condition arises in spastic spinal paraplegia, amyotrophic lateral sclerosis, cerebral hemorrhage, and disseminated sclerosis.

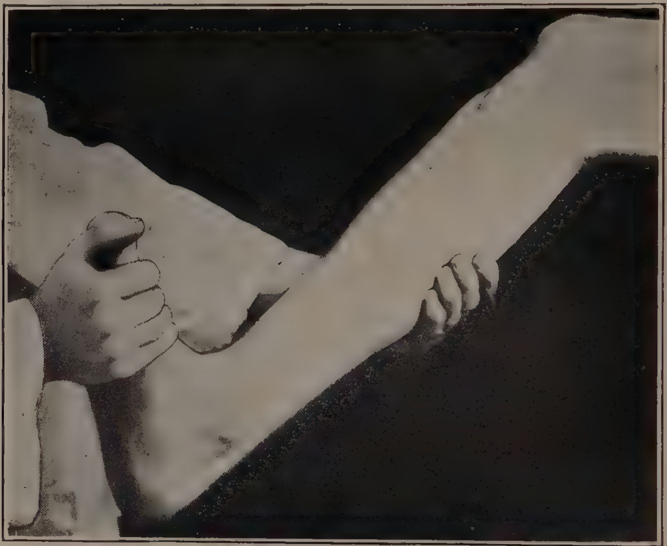


Fig. 240.—Elicitation of ankle clonus.

Abolition of the patellar tendon reflex is indicative of a break in the reflex arc due to disease of the sensory neurone, posterior root zone, or anterior root cells. Such lesions arise during the progress of tabes dorsalis, anterior poliomyelitis, peripheral neuritis, and trauma to the cord at the level of the reflex arc.

The Tendo Achillis Reflex.—This reflex may be elicited by directing the patient to kneel upon a chair, and, rendering the tendon taut by moderate dorsal flexion of the foot, the tendo Achillis is struck sharply, whereupon normally a sudden extension of the foot is produced. The reflex arc concerned in this reflex comprises the first and second sacral segments; and the significance of variations in the response is identical with those detailed under the knee-jerk.

The Jaw-Jerk.—In the presence of amyotrophic lateral sclerosis upon striking the point of the chin with the percussion hammer while the mouth is half opened the jaw-jerk is elicited. This reflex is distinctly pathologic and is not present in the normal subject. The reflex arc is represented by branches of the trigeminal nerve.

Ankle Clonus.—In eliciting this clonus the examiner grasps the calf of the leg in the palm of the left hand, while with his right hand he exerts pressure upon the fore part of the sole of the foot, thus maintaining the foot in a position of dorsal flexion. In the presence of disease of the upper motor neurone, as in disseminated sclerosis, cerebral hemorrhage, or spastic paraplegia, a series of regularly rhythmical contractions of the calf muscles ensue, which continue until the muscles are temporarily exhausted. The reflex arc in this instance is represented by the first, second, and third sacral segments.

Patellar Clonus.—The patellar clonus is elicited by placing the limb in a position of full extension and grasping the patella between the thumb and fingers and exerting strong downward pressure upon the quadriceps extensor tendon. In disease of the upper motor neurone a series of rhythmical contractions are set up in the quadriceps extensor analogous to that which is obtained in the case of the ankle clonus. The reflex arc in this instance is constituted by the second, third, and fourth lumbar segments of the spinal cord.

SECTION VIII

SPECIAL DIAGNOSTIC PROCEDURES

CHAPTER XXXII

MENSURATION, CYRTOMETRY, THORACENTESIS, AND VITAL CAPACITY

Thoracometry, or mensuration of the thorax, is employed to determine at consecutive examinations variations in the total circumference of the chest, to determine the presence of unilateral bulging or retraction of the thorax, and to estimate the total expansion of the chest.

In the determination of the total expansion of the thorax the difference between the circumference of the chest during complete expiration and during complete inspiration is taken, the difference between the two measurements taken at the level of the nipples indicating the total expansion of the thorax. A total expansion of three to four inches obtains in the average adult male subject, though slight discrepancies below or above these figures are not to be considered pathologic.

In the determination of unilateral variations in the size of the two sides of the thorax it is customary to measure from the midspinal line to the midsternal line upon each side and note any discrepancy in the two measurements. Allowance must be made for the fact that the right half of the thorax is normally slightly larger than is the left half. In making all measurements of the thorax the common tape measure is the appliance of choice, save in calculating the various diameters of the thorax, when a specially devised caliper is employed.

In determining the anteroposterior and transverse diameters of the thorax the caliper is used. In the estimation of the anteroposterior diameter one point of the instrument is placed over the midspinal line and the other over the midsternal line, and the measurement is read off on the scale of the instrument. The transverse diameter of the thorax is determined by applying a point of the caliper to each midaxillary line and reading the diameter as indicated upon the scale.

Mensuration of the abdomen is practiced in order to determine variations in the contour and extent of the abdominal walls in the presence of intraabdominal tumors, ascites, tympanites, ptosis, or cysts. Successive mensuration is practiced to determine any progressive enlargement of the abdomen from these conditions as well as to arrive at conclusions as to any diminution in these dimensions.

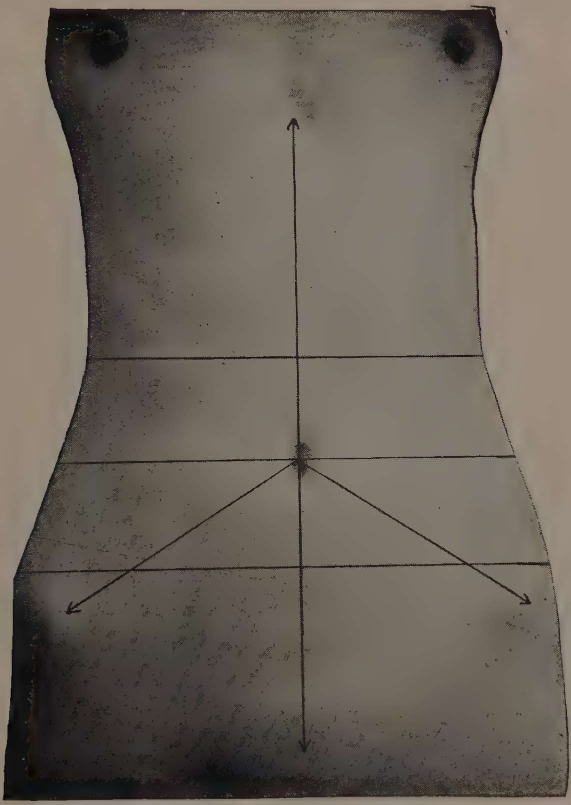


Fig. 241.—Showing the lines for mensuration. (From Crossen.)

In the practice of mensuration of the abdomen seven lines are employed; namely, (1) a line encircling the trunk at the level of the umbilicus; (2) a line encircling the trunk three inches above the umbilicus; (3) a line encircling the trunk three inches below the umbilicus; (4) a line extending from the umbilicus to the right anterior superior iliac spine; (5) a line extending from the umbilicus to the left anterior superior iliac spine; (6) a vertical line extending from the umbilicus to the tip of the ensiform process; and (7) a vertical line projected from the umbilicus to the symphysis pubis.

Cyrtometry, the determination of the curves of the surface of the thorax, is practiced by applying the cyrtometer accurately to the surface of the thorax. The cyrtometer consists of two pieces of flexible metal connected at one end by a hinge. In practicing cyrtometry of the thorax the hinge is placed over the midspinal line, and the blades of the instrument are accurately moulded to the surface of the thorax. Upon removal of the instrument a tracing may be made, showing the shape of a cross-section of the thorax, and revealing any unilateral variations of the two sides of the chest.

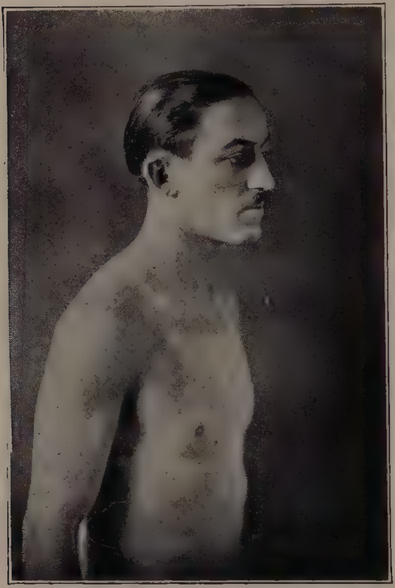


Fig. 242—Lateral site of election for exploratory puncture of pleura.

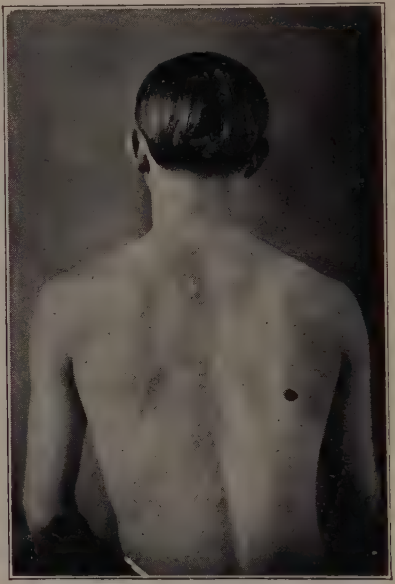


Fig. 243—Posterior site of election for exploratory puncture of pleura.

Thoracentesis, the aspiration of fluid from the pleural cavity, is usually preceded in practice by *exploratory puncture* of the pleural sac. The latter maneuver is frequently performed in order to establish the diagnosis of fluid in the pleural cavity and for the purpose of securing a specimen of fluid for microscopic and bacteriologic examination.

In the performance of the slight operation of exploratory puncture of the pleura, a large hypodermic syringe equipped with a heavy needle should be employed. Aseptic precautions should be strictly observed throughout the procedure in order to avoid the introduction of infective organisms into the pleural cavity, thus converting a serous into a purulent effusion. The pleural cavity may be entered in the scapular line in the eighth intercostal space or in the midaxillary line in the sixth interspace with

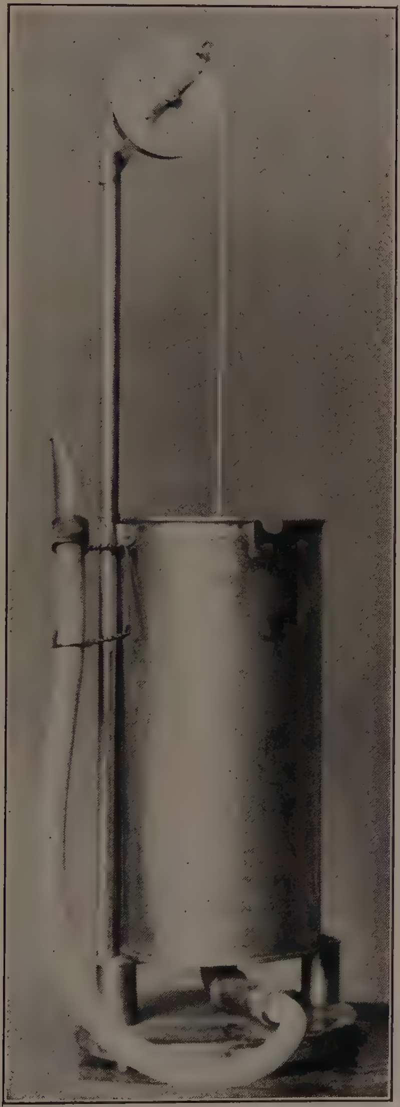


Fig. 244.—Simple spirometer for measuring the vital capacity of the lungs.

equally satisfactory results. After cleansing the skin it may be frozen with ethyl chloride but usually this is not necessary. Elaborate preparation with cocaine injection is unnecessary and usually causes as much pain as the puncture. When all is prepared, the needle is grasped firmly, the base resting in the palm of the hand. The needle is inserted by a strong steady push, care being taken to note the consistency of the tissues through which it passes. The grating feel of a thickened pleura, the lack of resistance when fluid or air is encountered, and the peculiar rubbery feel of the intact lung are recognized only after having experienced the sensations. In securing a specimen by exploratory puncture, a few cubic centimeters should be withdrawn, and immediately transferred to a sterile test tube for microscopic and bacteriologic study.

Vital Capacity of the Lungs.—The vital capacity of the lungs is the amount of air which can be expelled after a forced inspiration. The vital capacity is measured by means of an ordinary spirometer of seven or eight liters capacity. The subject is allowed three trials and the maximum reading is recorded. The amount of air expelled by the adult consists of the ordinary tidal air which amounts to about 500 c.c., plus complemental air 1500 c.c., and supplemental air 1500 c.c. These amounts may vary considerably with age, sex, stature and training in chest expansion. Smiley and Augustine found that the vital capacity of the Negro was less than that of the white race. The area of body surface has been found a fairly reliable standard for different individuals. Diseases of the lungs, such as pneumonia and tuberculosis, cause marked diminution of vital capacity. Severe heart disease produces a similar diminution. Sturges and Peabody and Head and Smith, by means of studies of vital capacity, have shown that the maximum respiratory rate which allows for respiratory efficiency is about 35 per minute, thus clearing up some of the problems of dyspnea.

CHAPTER XXXIII

RADIOLOGY IN PHYSICAL DIAGNOSIS

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The ever-widening field of application of the x-ray in diagnosis has developed radiology as a branch of medicine *sui generis*. No other proof of its importance need be adduced than the fact that fifty per cent or more of hospitalized patients find their way at some time or other to the x-ray laboratory. The percentage of subjects to whom such procedures are applied probably exceeds that of those receiving any other single diagnostic measure. It is therefore evident that the student should have some knowledge of this diagnostic branch, but it is difficult to determine how much is essential to a course in physical diagnosis for medical students in their earlier years. This chapter is an attempt to present for the consideration of the student the fundamental elements underlying radiologic examination. Every effort is made to avoid the confusion likely to arise from excess of detail. More specific information as to diagnostic radiologic measures should come in the later years of the curriculum, preferably in the fourth year of the medical course. At that period systematic instruction in radiology should be imparted, preferably by the case method of instruction.

As will appear below, radiologic examination is in a very real sense a method of indirect visualization. For purposes of convenience it may therefore be considered an extension of the method of *inspection* as that term is understood in physical examination. The term "inspection" is quite rigidly limited to external surfaces, but should include those cavities which may be considered external surfaces because they can be inspected. In contrast to inspection, radiologic examination visualizes structures, organs, and tissues situated far below the surface. This visualization is not so complete or so full as actual inspection of the structures, organs, or tissues would be were they directly under the eye.

Medical students of the present day have had in the premedical course more or less instruction in physics and have some knowledge of radiations in general, and of x-rays and radium rays in particular. Nevertheless, for the purpose of refreshing the memory, a few elementary points in regard to radiation phenomena should be introduced.

X-rays originate at the point of arrest of moving electrons. In order that an electron be set in motion it must be dissociated from its parent substance by high electrical tension. To secure the emission and necessary rapidity of movement, electrons have to originate in a gaseous medium, in a pressure far below that of the atmosphere. The production of x-rays therefore requires:

(1) a high tension electrical current which
is unidirectional
acting on
(2) two electrodes functioning in a
partial vacuum.

These conditions are fulfilled by an x-ray tube activated by the so-called x-ray transformer.

The power to penetrate substances opaque to ordinary light is one of the most important properties of the x-ray and constitutes the basis of its medical application. This power of penetration is in proportion to the density of the matter interposed in the path of a beam of x-rays. The degree of penetration is ascertainable by means of two other important qualities of x-rays which are their power to excite fluorescence in certain salts and to reduce the silver in photographic emulsion. The degree to which both of these phenomena are brought about is directly dependent upon the intensity of the incident radiation. Intensity of radiation is the quantity of radiation reaching a unit area in a unit of time.

In order that x-rays may be useful, it is necessary that there be some means of ascertaining their penetrative powers. This end is secured by means of the properties just mentioned above. There are hence two methods of making such observations, the photographic and the fluorescent. In the case of the first named, the photographic materials used differ little from those employed in ordinary photography. However, the photographic plate (of other days) or film (of today) is enclosed in an envelope or other suitable holder opaque to daylight and the holder and film are both exposed; since the x-light will penetrate the envelope or holder, an image can be produced. Though this is a photographic method, the resultant image is not a photograph, since it is not produced by visible light, nor are any lenses employed.

To record the fluorescent effects of x-rays, the necessary salts also require special treatment. They are spread on and caused to adhere to a material easily penetrable by the x-rays, and having a flat surface. The material fulfilling these requirements is cardboard. When so prepared and suitably mounted and refined, such an instrument is known as a fluorescent or fluoroscopic screen. The light given off by such a screen is not x- but visible light. It is very faint and hence it is necessary that it be used in the dark. The exact nature of the salts used in the composition of the fluoroscopic screen need not concern us here, since this is a branch (as, in truth, are all matters of x-ray apparatus, equipment, etc.) that properly belongs in the field of x-ray technology.

Some general remarks on apparatus, however, are in order. The x-ray transformer has been mentioned. This piece of apparatus supplies the x-ray tube with the electrical energy at the proper tension. They are merely "step-up" transformers equipped with a rectifying device which gives a unidirectional current. As the latter is of high tension and capable of sparking several inches off its lines, it must be conveyed to the x-ray tube under conditions which eliminate the possibility of such an occurrence. Electrical shocks are ever a potential danger in the course of x-ray procedures. The high tension wiring used in connection with x-ray apparatus is more complex in appearance than it is in reality.

The x-ray tube, in addition to the fragility inherent in a piece of glass apparatus, is when in operation charged with high tension electricity, which has to be guarded against just as in the case of the high tension lines mentioned in the preceding paragraph. X-ray tubes in action emit rays from most of their parts. These "wild" rays, besides being useless for the purpose of examination, are a serious menace to both operator and patient and must be suppressed. It is therefore necessary that the x-ray tube be mounted in such a way as to guard against breakage and the possibility of electrical shock and useless ray emission. These needs are fulfilled by a tube stand or tube holder, which is mechanically designed to secure the necessary flexibility in operation. When a fluoroscopic screen is mounted in connection with the tube stand, the apparatus is known as a fluoroscope. If the x-ray tube is mounted beneath a table and there is a fluoroscopic screen fixed above it, the apparatus is called a trochoscope.

Fluoroscopy and radiography do not accomplish the same ends; they should be considered as complementing one another. Since each has its own distinct advantages, one should never rely on either as a method of examination to the exclusion of the other. Radiography may be depended upon to produce a more sharply defined image than can be obtained by fluoroscopy. In addition to being a more delicate record, it forms a permanent one. On the other hand, the chief point in favor of the fluoroscopic as compared with the radiographic examination lies in the fact that the fluoroscope determines the existence and nature of motion in organs where movement is found. The reasons for these respective advantages are clear when the differences in the purpose of the two methods are understood.

The image observed on a fluoroscopic screen is the product of the average of a discontinuous emission of fluorescence due to the pulsating electric current delivered to the x-ray tube. This means that there is a flickering of the light from the screen. It is impossible to attain a continuous and steady fluorescence. Moreover, only a limited amount of x-ray can be applied in a fluoroscopic examination because of the risk of excessive exposure of the operator and patient to x-ray. This contributes greatly to the faintness of the image seen on the fluoroscopic screen. On the other hand, when radiographs are examined, images are visualized because of the intense light which is transmitted through the film. Such intensity of light cannot possibly be had from the fluoroscopic screen. Were it possible to attain a comparable intensity on the screen, the amount of exposure of both the patient and operator to the x-rays would be exceedingly dangerous. The light passing through radiographs when they are examined with suitable illumination will always be greater in quantity than that originating from a fluoroscopic screen. Clear definition, both radiographic and fluoroscopic, is a function of light quantity; hence, the radiograph is always superior to the screen image in respect to clear definition. Measured in radiographic terms, the screen image can be regarded as of the value of an inferior radiograph.

The chief difficulty in radiographic examination is that the desired results can be attained only where absolute immobility is secured; therefore, there are certain conditions in which fluoroscopy is the only practical method of radiologic examination. With such exceptions, the result obtained by fluoroscopy can be duplicated with greater ease and less risk to patient and operator by radiography. The resultant findings are much more precise from the standpoint of diagnosis and more desirable in view of the fact that they form a permanent record. A commonly encountered error in regard to fluoroscopy usually advanced by the inexperienced is the statement that conditions can be better observed on the fluoroscopic screen than by radiography. Those having the greatest experience with x-ray examination in all its phases would probably agree that the chief function of the fluoroscopic examination is to enable one to do more accurate radiography. For example, the combination of fluoroscopy and radiography in the examination of the respiratory and alimentary tracts is absolutely essential if the best results are to be attained.

Since x-rays are absorbed by the medium through which they pass, and the rate of absorption is proportional to the density of the medium, the chief problem is to discover the density variation in the human organism which renders the rays diagnostically useful. For purposes of simple exposition, it may be said that there is a base line of density in the human organism. This base line is made up of those organs and tissues which closely approximate each other in specific gravity which property is more or less largely determined by their content of water. There may be among them a fair degree of difference in density as determined by more delicate methods than the measure of this quality by the x-ray, but for radiologic purposes this difference is negligible.

This base line is made up of all the parenchymatous organs, muscle, adipose tissue, integuments, nerve, vessels and their contents and the water in the tissues themselves and in the organs which may contain it. (Fig. 245.) It also includes ingested food. Above this common base line in which the density of the tissues is so nearly equal that there can be virtually no difference in their absorption of x-rays, can be placed those structures with a greater density. These contain mineral salts, chiefly calcium; the most important of these structures are the skeleton and the teeth. Below the base line are the substances of lesser density than those of the base line. This class is the one which includes organs containing air or any other gas.

Upon practical application, it is found that some of the organs and tissues which are included in the base line are occasionally delineated from surrounding tissues which have practically the same density. For example, the kidney is usually outlined on radiographic examination. This constitutes a special case, however, for it is outlined because it lies in its own fatty capsule.

There is sufficient difference in density between the parenchyma of the kidney and its fatty capsule to render visible the outline of this organ. Likewise, the lower border of the liver and the outline of the spleen are occasionally demarcated from the other abdominal organs, and at times the bodies of the muscles of the extremities are clearly and beautifully outlined. There is so much inconstancy of delineation, both of the viscera and of the

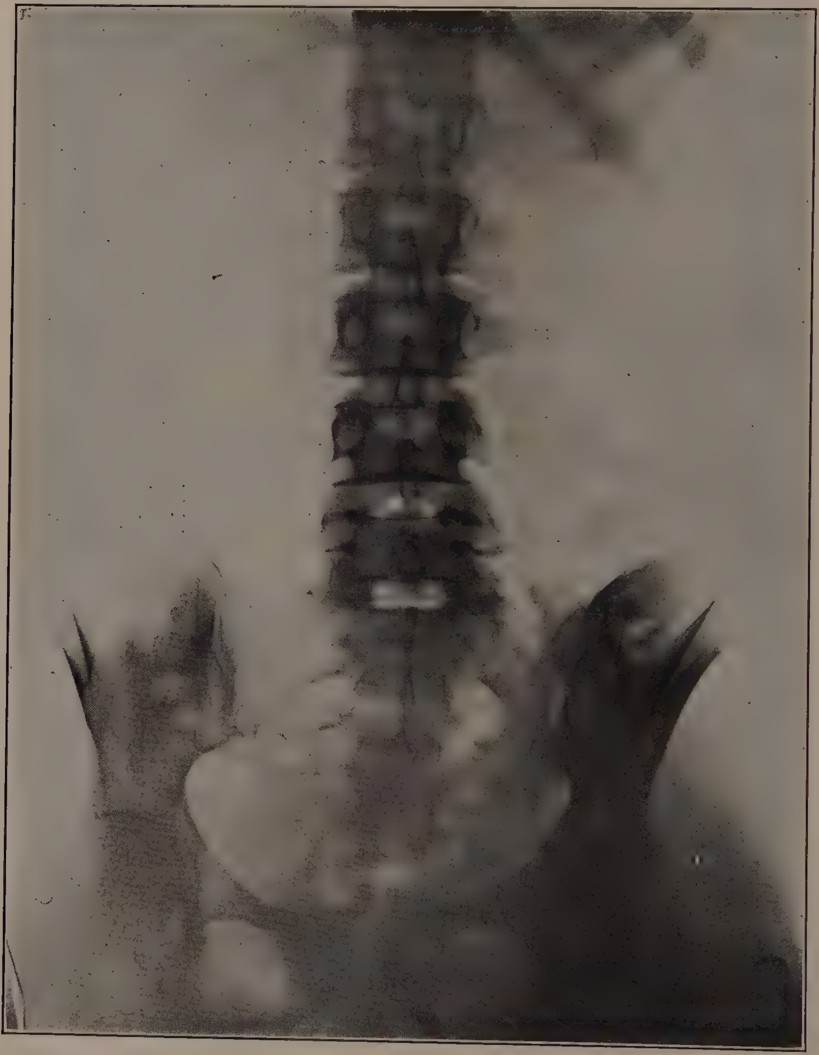

Fig. 245.—Radiograph of the abdomen. Only the bones are visualized, the parenchymatous organs and soft tissues of the abdominal cavity are not differentiated from each other.

muscles, that, as a method of study, this visualization is of very little practical consequence. For example, a muscle body is probably visualized because it is outlined by the less dense fatty tissue separating it from adjacent muscles. When the outlines of parenchymatous organs are clearly demonstrable, the question arises as to whether they are normal or have increased in density through a pathologic process. It is probable that in many cases the outlines of normal organs are shown in the absence of increase of density.

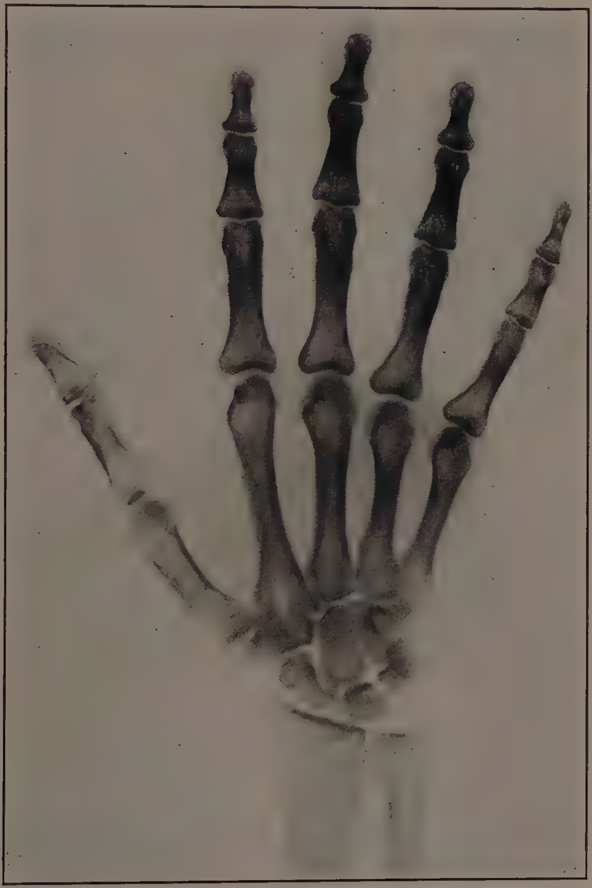

Fig. 246.—Film of normal wrist and hand revealing bony structure only. The soft tissues are outlined to a slight degree.

Above the base line of the general density found in the human body occur those organs, tissues, and structures which normally contain calcium and other mineral matter (Fig. 246). These are,

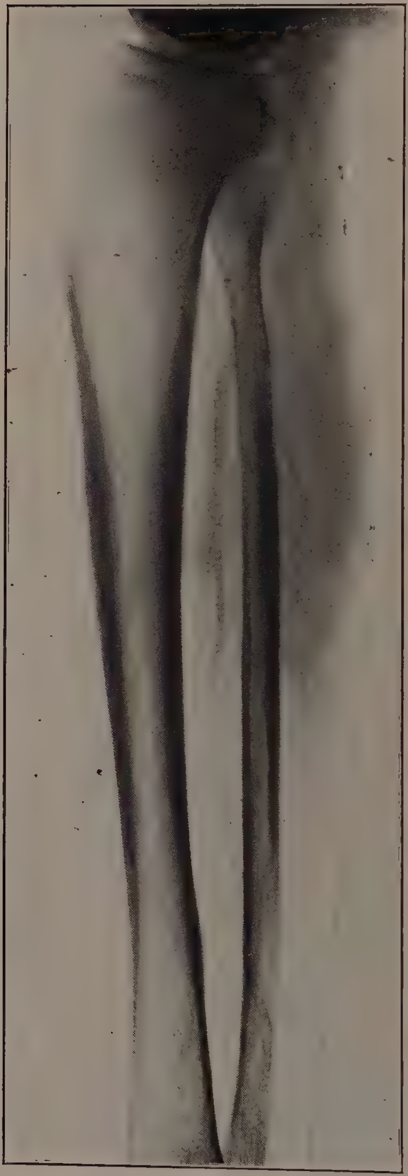

Fig. 247.—Film of the leg, showing extreme degree of calcification of the arteries. This is too intense to be considered as physiologic. The student will note that both the bones and arteries are made visible by the calcium which they contain.

of course, chiefly the skeletal and dental systems. In certain cases and certain localities, calcification and ossification other than in these two systems may be encountered and should be considered as normal. For example, some degree of calcification of blood vessels, especially of the arteries (Fig. 247), is so often present in those of advancing years that it may be considered physiologic and only indicative of aging tissue. It should be noted that this process is one of calcification and not of ossification. Another example of calcification which is physiologic and therefore a nor-

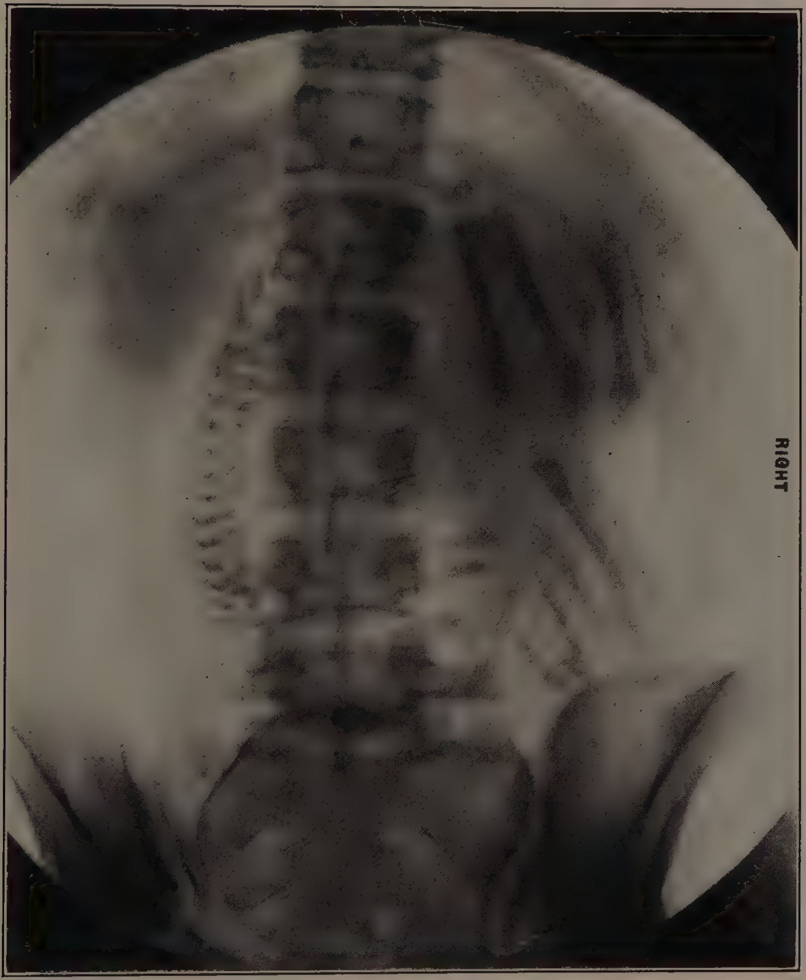

Fig. 248.—Radiograph of abdomen in pregnancy, with the ossified fetal skeleton shown.

mal process, is the deposition of calcium in the pineal body, which is observed in the majority of films of the skulls of adult patients. Certain sorts of calcifications should also be considered physiologic because of their wide prevalence. Examples of this are the calcifications of the costal and laryngeal cartilages in the majority of adults. Of like nature is ossification of the fetal skeleton in utero (Fig. 248). Occasionally plaques of normal bone are found in the falx cerebri. These are to be considered physiologic or, in any case, not pathologic. The above summary approximately includes the normal or physiologic instances in which the density is increased so as to become greater than that of the general base line of the parenchymatous and soft tissues.

In discussing the structures above the base line of even density, that is, those which contain calcium and other mineral matter, discussion of the teeth will be omitted as this properly lies in the field of dental surgery. For reasons which are obvious, vascular calcification, pineal calcification, and other normal physiologic findings of calcified material will also be disregarded and the discussion will be confined to the skeleton.

Bone, because it is of greater density than the tissues which surround it, was the first human tissue satisfactorily examined by x-ray procedures. The quantity of calcium present in bone, however, is variable and is dependent under physiologic conditions, on such factors as age, sex, muscular development, activity, and diet. Even in pathologic conditions, the quantity of calcium in the skeleton may vary in what might be called a physiologic range. The calcium content of bone, as gauged by its rate of absorption of x-rays, may be shown to vary either because of the relatively small size of the bone, or because of the actual diminution of the calcium which it contains. In other words, the quantity of calcium which is present may be small either because the bone is small in size but normal in calcium, or because it is normal in size but has a diminished calcium content. Both of these factors have a bearing on the amount of x-ray that can be absorbed, or, to express it differently, are sources of variation in density.

It is all too commonly supposed that bone is a fixed tissue and remains so throughout life. Nothing could be more erroneous than such a belief, for this tissue is very labile and probably ranks higher in instability than any of the other tissues of the mesenchyme. Bone cannot be considered to have reached its full development until the last epiphyses have united. From infancy to that period, there is more or less lack of calcification in the skeleton. The uncalcified portions, usually epiphyseal cartilages, approximate the base line of the general density of the soft tissues of the human body.

The bones of the female are smaller and lighter and possibly contain less calcium than the bones of the male. Though this difference is not measurable, it is probable that the skeleton of an adult in full vigor contains more calcium than in the earlier years of life. Again, in old age, the bones tend to have less and less calcium and to decrease appreciably in density. This change, however, is not to be considered pathologic. The rarefaction is possibly to be attributed as much to decreased activity as to other senescent changes. It should be observed that the strong and muscular individual will have a larger bone with a greater calcium content than the individual of lesser development.

Below the base line are those tissues which have a lesser density than the soft tissues of the body because of their content of air or other gas. This, of course, primarily includes the respiratory system and its accessory or adjunct parts. In this class are the nasal passages, the nasal accessory sinuses, the pharynx, the mastoid bones, the larynx, the trachea, the bronchi, and the lungs. In addition, gas, probably air, is practically always present in some quantity in the stomach; in the fundus of the stomach, if the individual is in an erect position. It is then noted just below the left leaf of the diaphragm. This bubble of air shifts as the individual shifts position. Gas of some nature is vicariously present in other portions of the gastrointestinal tract; however, air is generally confined to the respiratory system, and the physiologic appearance of air or other gases observed elsewhere, as for example, in the alimentary tract, is of little or no import. For this reason, the physiologic aspect of the air-containing structures of the respiratory tract merits attention.

All three of the densities of the human body are encountered in the upper portion of the respiratory tract; for example, the nasal passages and their accessory sinuses (Fig. 249), when subjected to x-ray examination, bring out all three of these densities. Air, soft tissue, and bone are all present, each contrasted against the others. Because of this natural contrast in the case of these structures, their size, position, contours, boundaries, and relations are well defined. Similarly, the pneumatic structure of the mastoid portion of the temporal bone (Fig. 250) is, on a smaller scale, advantageously examined by means of the x-ray.

The column of air ordinarily extending from the oral cavity and the pharynx to the bifurcation of the trachea constitutes an example of air seen in contrast to soft tissue only. Due to this contrast, the size, shape, capacity, and position of both the nasal and laryngeal pharynx, larynx, and trachea can be normally determined to some extent.

It is within the thorax that the respiratory system is most suitable for radiographic and fluoroscopic examination, and that most findings are secured. This is the location par excellence for radiologic examination to come to the aid of, and to supplement, the older means of physical diagnosis. Because of the air contained in the lungs (Fig. 251), the enclosing soft tissues and the bony portions of the thorax are brought into contrast with each other. The bony elements of the thorax can be omitted from this discussion because they can be adequately examined by x-ray method regardless of pulmonary aeration. The chief value of radiologic examination of the thorax lies in the fact that tissues belonging to the base line of equal density can be deline-

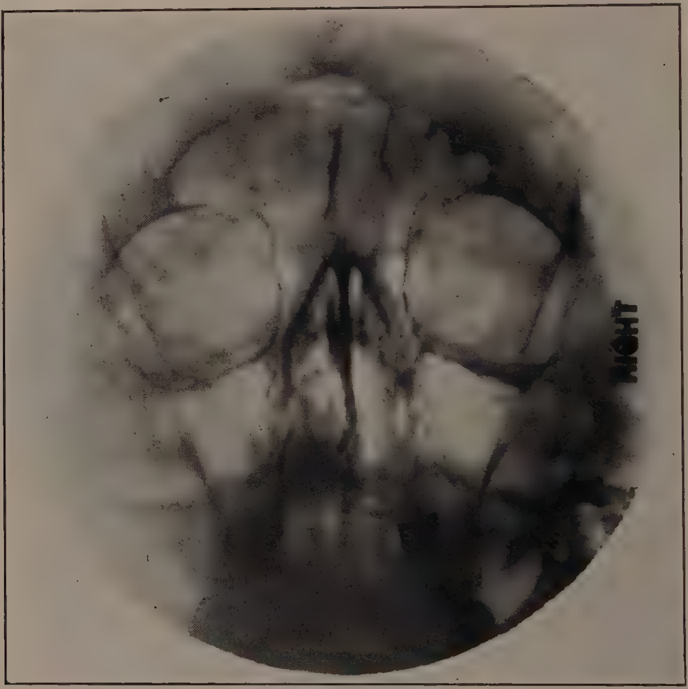

Fig. 249.—Radiograph of skull demonstrating air-filled frontal sinuses, ethmoid cells, nasal passages, and maxillary antra.

ated to some extent. The bases of the lungs outline the upper surface of the diaphragm on each side. Their medial aspects map out the heart, and to a lesser but still important degree, the great vessels of the mediastinum. The aerated lung normally brings into contrast the dense structures at the root of the lung, such as the major bronchial tubes and their accompanying vessels, the lymph nodes, and the supporting tissues. The branches of the bronchial tree, with their accompanying structures, are also made visible from the root toward the periphery of the lung until these branches have diminished in size to a certain unknown point where they are too fine to be seen in the great mass of air-containing lung tissue. The soft tissue of the thoracic walls is shown in contrast both to the ribs and spine already referred to, and to such tissues as are adjacent to them. This is also true of the soft tissues lying above and internal to the apex of the lung at the base of the neck. The pleura, although it may be brought into contrast with the lung in pathologic conditions, is not normally visible.

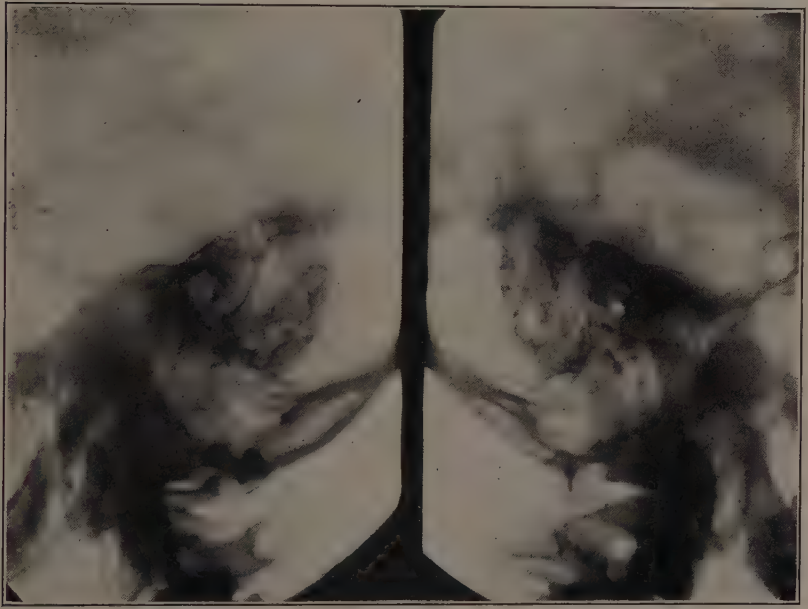

Fig. 250.—Radiograph of mastoid portions of temporal bones demonstrating the air-filled mastoid cells contrasted with the enclosing bony septa.

The presence of foreign bodies in the body properly belongs in the section on pathology instead of radiology. They can be dealt with at this point to better advantage to the student. In order to be discovered, foreign bodies must vary in density from their surroundings to a considerable extent. If they approximate the specific gravity of their surroundings, there is no possibility

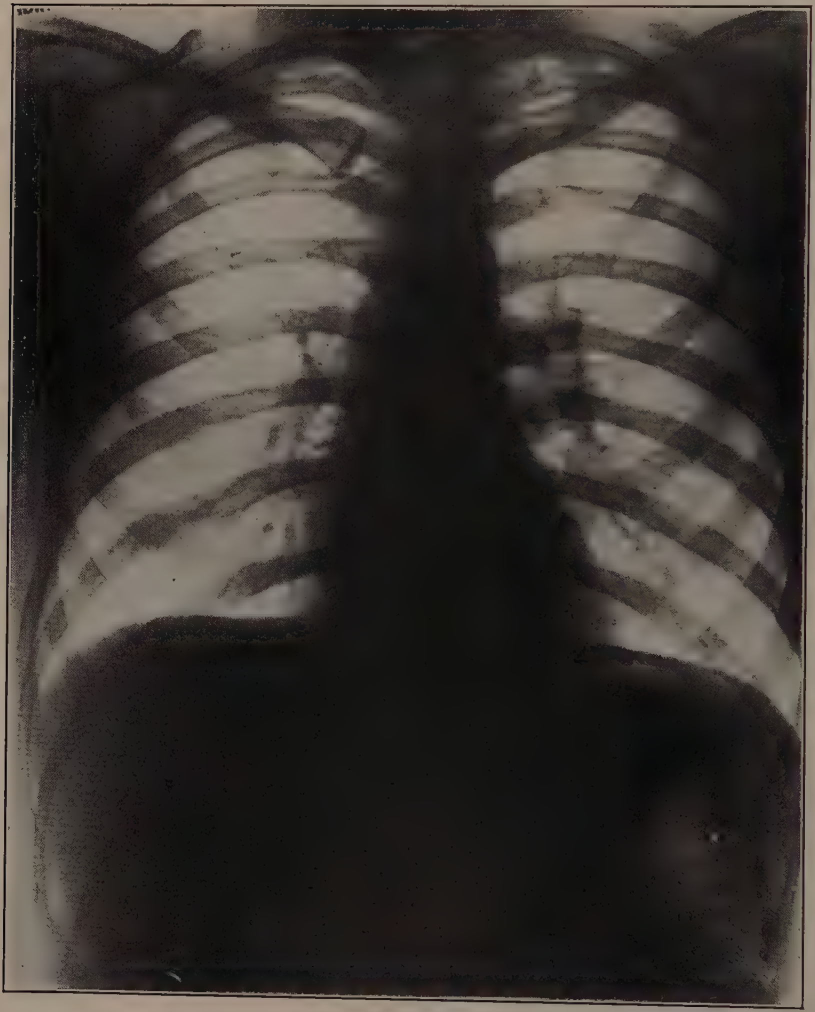

Fig. 251.—Radiograph of chest. Note that both bone and soft tissues are in strong contrast with the air-containing lungs.

of distinguishing them; hence they cannot be visualized. Discoverable foreign bodies are chiefly metallic; these can be observed in surroundings as dense as bone, just as metallic fillings in teeth are visualized (Figs. 252 and 253).

To summarize, it may be said that the great bulk of tissues in the human body are so nearly alike in density that there is no appreciable difference in their rate of absorption when these structures are exposed to the x-ray. A few tissues, which contain a large percentage of mineral salts, are of a greater density and therefore absorb more x-rays so that they are visible in contrast with the structures which are of the average base line density. The chief examples of this class are the skeletal and dental systems. On the other hand, there are organs which are less dense than those on the general base line. These are structures which contain air or other gas. Excepting small amounts of gas which are physiologically present in many organs, the respiratory system is by far the most important in this class.

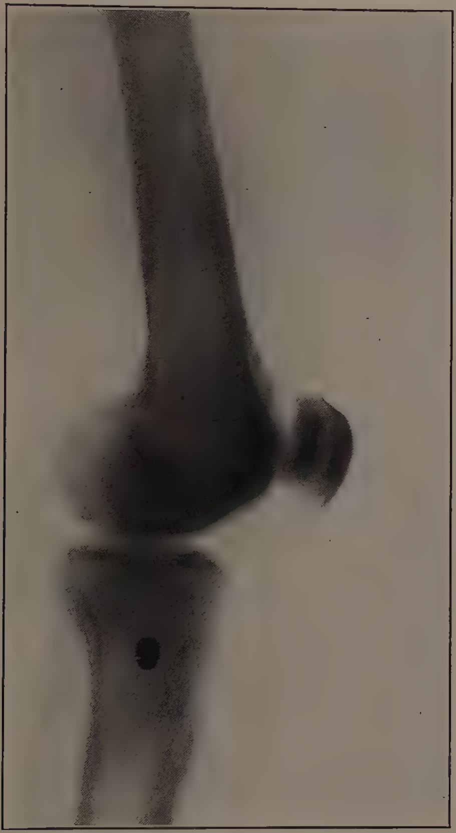

Fig. 252.—Lead bullet overlying tibia. Note strong contrast between the bone and the bullet. This is due to the greater density of the latter.

Since these two classes are exceptions to the general rule, the great majority of human tissues are on the general base line of approximately the same density, it may be readily seen that if radiologic examinations were solely dependent upon variations physiologically present, the utility of this mode of study of the human organism would be very limited in scope.

From the very early days of the development of medical radiology, this defect in the method was apparent. To increase the efficacy of the x-ray as a means of diagnosis, attempts were made to create or enhance contrasts in density. The more important methods of generating artificial contrasts, which resulted from such efforts, will be mentioned in chronological order.

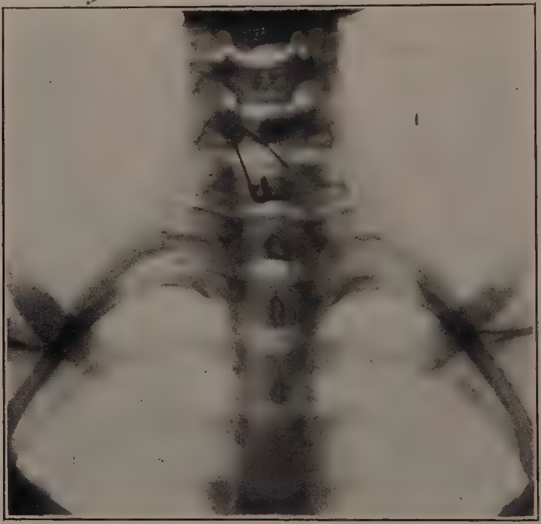

Fig. 253.—Radiograph of foreign body, safety pin, in the trachea. It is in contrast with the spine and the column of air in the trachea.

The first attempt in this direction consisted in injecting into sinuses or fistulous tracts, such as those of deep suppurating wounds, material with a high specific gravity. Originally a bismuth preparation known as bismuth paste was used for this purpose. Because of the opacity of the material which had been forced into it, the outline of such a fistulous tract or sinus was at once rendered visible and its course could be traced. Soon after this, a satisfactory method was evolved whereby the ingestion of opaque material in the alimentary tract would produce satisfactory conditions for fluoroscopic, and later for radiographic, examination. This procedure is known as gastrointestinal fluoroscopic, radioscopic, or radiographic examination. A considerable time after this, pyelography, the study of the kidney pelvis after the injection of an opaque medium into its hollow portions, was successfully carried out. The images thus obtained are known as pyelograms.

It will be observed that up to this point the efforts to generate artificial contrast were confined to producing an increased density to contrast with a lesser one. The next step in development was from the opposite approach, that is, an attempt was made to create a contrast with substances found below the base line of uniform density referred to above. This end was secured by the injection of air or other suitable gas into the peritoneal cavity, either directly or via the vaginal and uterine canals, the procedure being known as pneumoperitoneum. Following up the evolution of this type of procedure, experiments were made in pneumoventriculography, which consists of injecting air into the ventricles of the brain after aspiration of the necessary amount of cerebrospinal fluid to permit ingress of the air. An addition to this procedure is myelography, that is, the injection of air into the spinal canal by lumbar puncture.

The next development in the production of artificial contrast media once more employed the class of materials lying above the base line of even density, material of greater density being placed in a hollow cavity for purposes of studying its physical characteristics. In this case, the spinal canal was the cavity studied. The medium injected therein was iodized oil which has a heavy iodine content and is quite opaque to the x-rays. Iodized oil delineates the cavity of the spinal canal to some extent, and, more important, establishes its patency.

Iodized oil was in a very short time also used for purposes of exploring the respiratory tract, first in its lower portion, the bronchial tree (bronchography) and subsequently for exploring the nasal accessory sinuses (sinus mapping). At approximately the same time a method similar to the preceding was also applied to the study of the uterine cavity and the fallopian tubes by direct injection via the cervical canal. This is known as hysterosalpingography.

The next development of artificial contrast media was in cholecystography. In this, a suitable substance excreted by the liver with the bile reaches the gall bladder and is concentrated there. This substance can be administered either by way of the circulation or by oral ingestion. After a certain lapse of time, it is so concentrated in the gall bladder that sufficient density is attained to make the organ visible and to produce adequate images which are known as cholecystograms.

The methods described above vary in their value in radiologic diagnosis. The following explanations are intended to give the student some knowledge of the results which may be obtained by artificially producing contrasts in density, and to suggest which of the methods are most useful and most commonly applied.

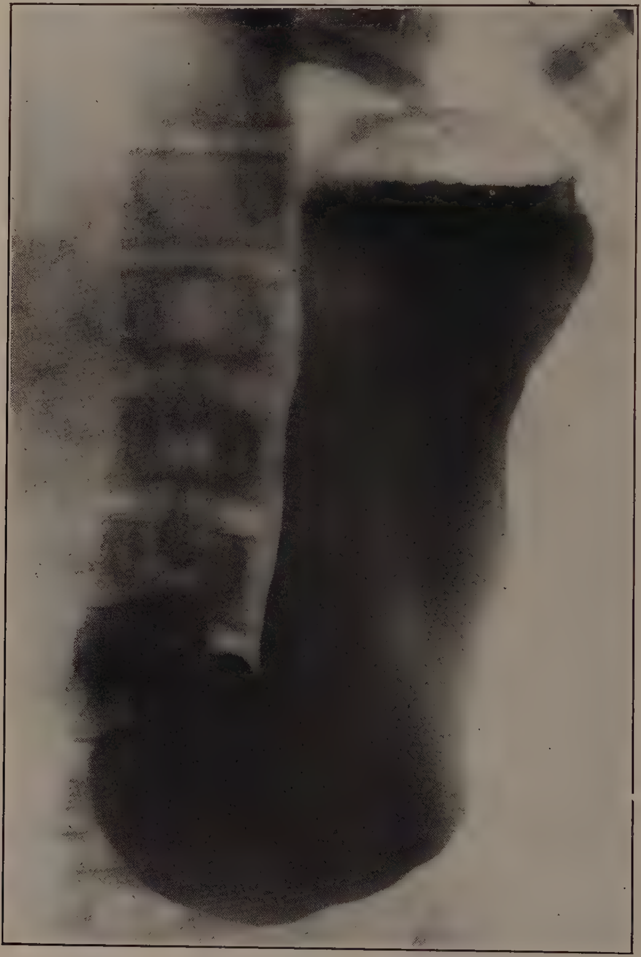

Fig. 254.—Radiograph of barium-filled stomach.

The opaque meal, as shown by fluoroscopic and radiographic examination, gives information as to shape, size, position, relation and contour of certain segments of the alimentary tube. The most valuable results from such an examination are obtained in the cases of the stomach and duodenum (Fig. 254). Of somewhat less value is such an examination of the esophagus (Fig. 255). In the case of the jejunum and ileum, little is to be learned. Information as to the physiologic condition, location, and other characteristics of the appendix may also be ascertained in this manner. In the study of the colon both the opaque meal and the barium enema (Fig. 256) are of great value, and both should be used. The radiologic gastrointestinal field is so great that it cannot be dealt with in a work on physical diagnosis.

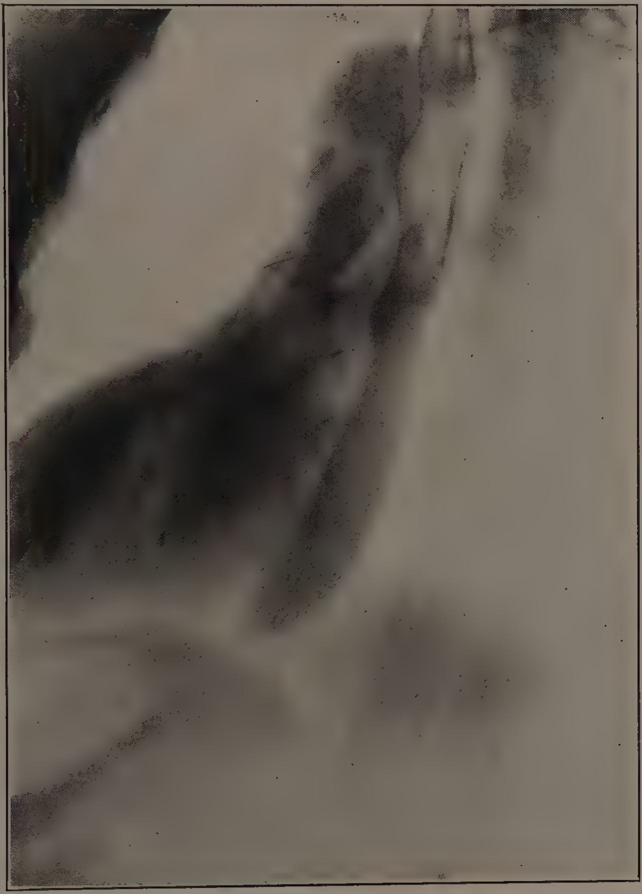

Fig. 255.—Radiograph of esophagus filled with barium. It is viewed obliquely. Outline of the heart lies anteriorly; the spinal column posteriorly.

In the urinary tract (Figs. 257 and 258), the size, position, relationship, contour, and capacity of the several divisions of the tract are advantageously studied by the methods named.

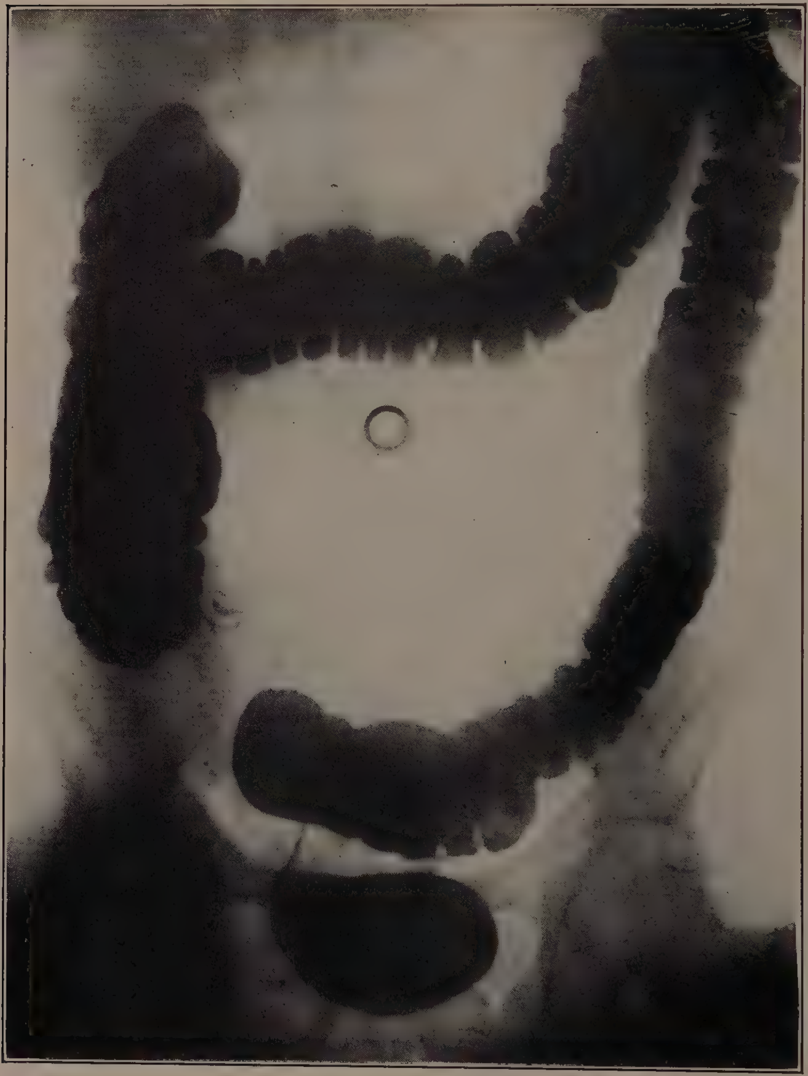

Fig. 256.—Radiograph of colon filled with barium enema. Note the barium-filled appendix.

In contrast to the preceding, the injection of air, a substance less dense than that of the general base line, into the peritoneal cavity (Fig. 259) brings into visualization the outlines of the solid structures contained in the abdominal cavity. It reveals, however, little comparable to the qualities which are disclosed when

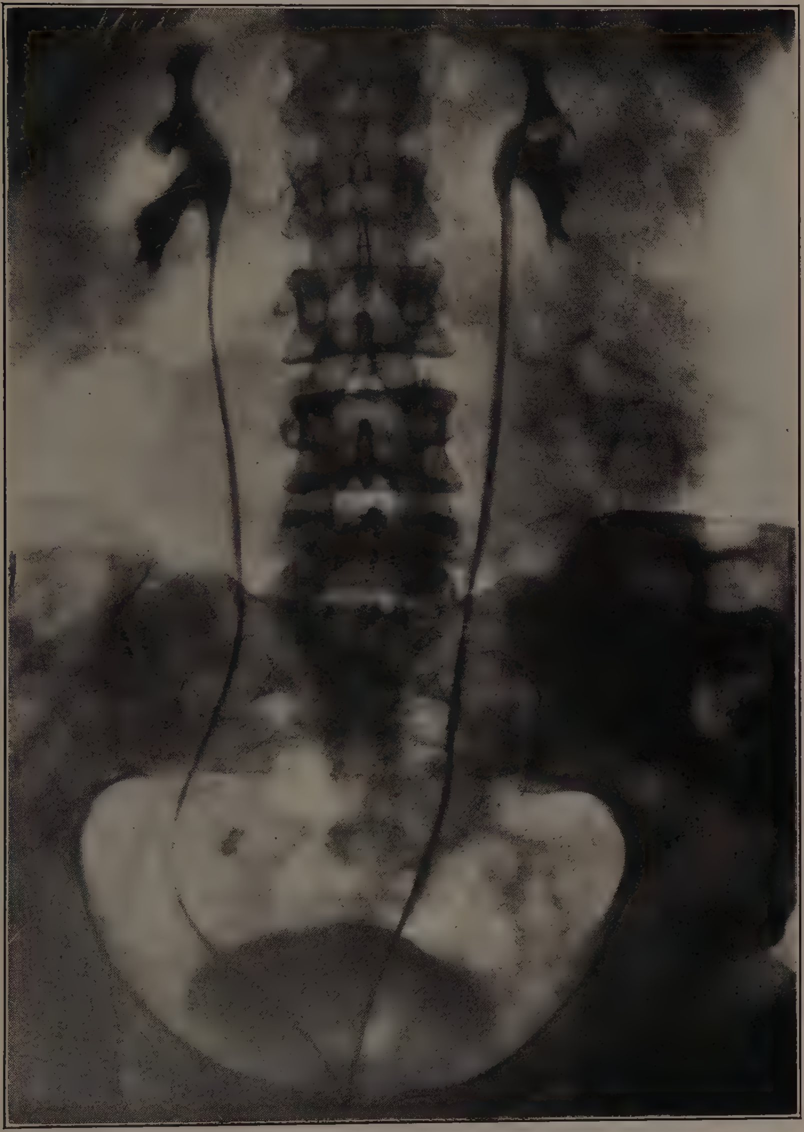

Fig. 257.—Pyelogram. The course of the ureters is shown by the opaque catheters which they contain.

a hollow viscus is filled with dense material. Gas in the peritoneal cavity can shift from place to place on change of position of the body; hence it gives a certain amount of contrast throughout the abdominal cavity. The procedure has proved itself of little practical value and has been largely abandoned.

In the cerebrospinal system, on the other hand, the injection of air into the ventricles of the brain and subarachnoid space and spinal canal has been quite valuable in showing the capacity, outline, relationship, position, form, and shape, of the ventricles and subarachnoid space. (Figs. 260 and 261.) Patency of the spinal canal can be established by this means. The injection of air (aerography) into the cerebrospinal cavities has been diagnostically superior to pneumoperitoneum. By this means, dilatations, occlusions, displacements, and other deformities can be shown in many cases in which the usual diagnostic measures have failed.

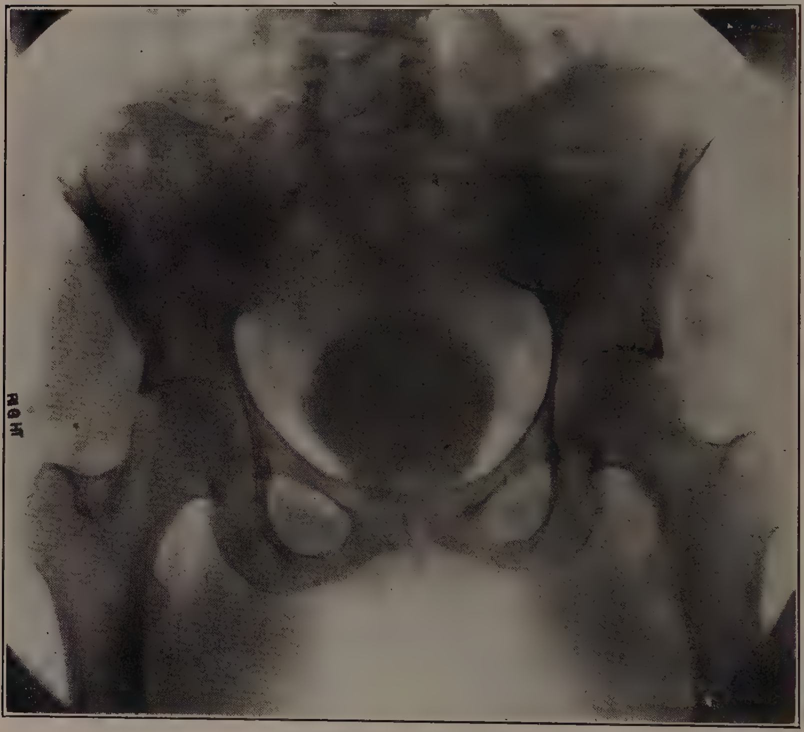

Fig. 258.—Cystogram revealing size, contour and capacity of the urinary bladder.

Aerography is frequently employed to great advantage in the serous cavities; however, it has not proved very useful in the study of joint cavities, and the same may be said of diagnostic

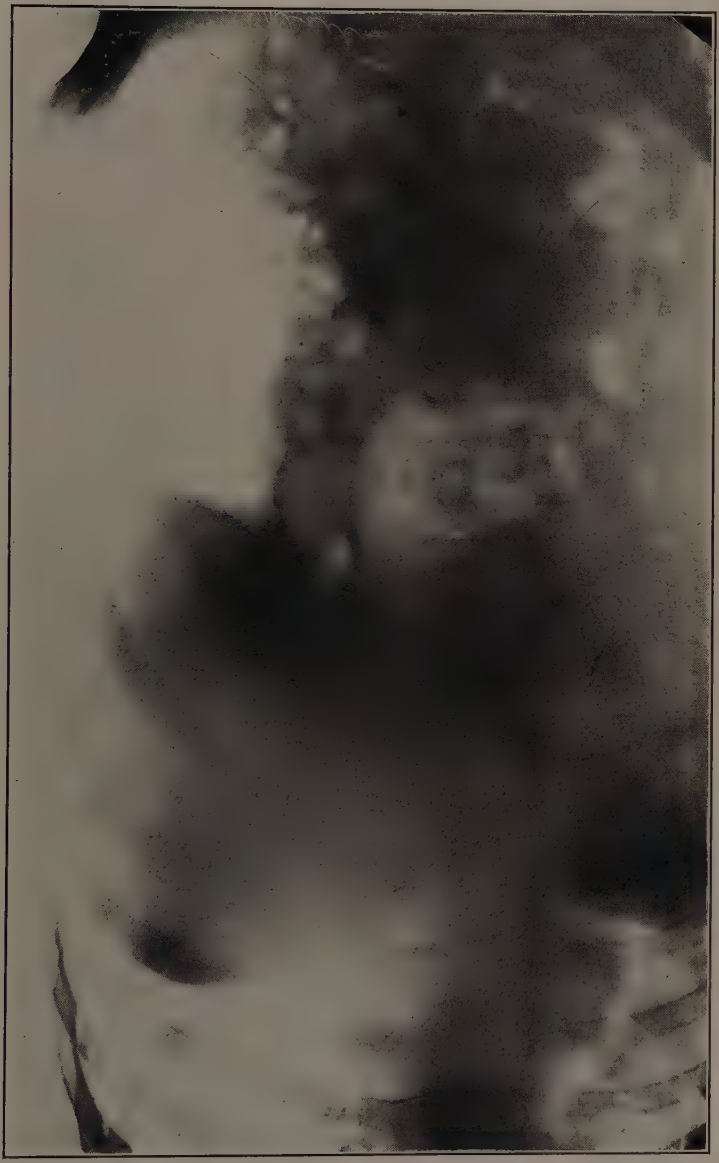

Fig. 259.—Pneumoperitoneum, patient lying on left side. The light area in the right flank is air in the peritoneal cavity.

pneumothorax. On the whole, the most valuable employment of air injection, as is seen above, is for pneumoventriculography.

Iodized oil, originally intended for exploration of the spinal canal (Fig. 262), has been found far more useful in the study of the respiratory tract and the uterine cavity. When instilled into the bronchial tree by way of the trachea (Fig. 263), it reveals the condition of this structure. Thickening, dilatation, relationship, the nature of dense areas in the lungs, and similar findings which would not otherwise be determined are thus discovered. When instilled into the upper respiratory tract, it will reveal to some extent the capacity, outline, and shape, especially on change of position, of the nasal accessory sinuses (Figs. 264 and 265). It has not been found useful in the study of the mastoid portion of the temporal bone.

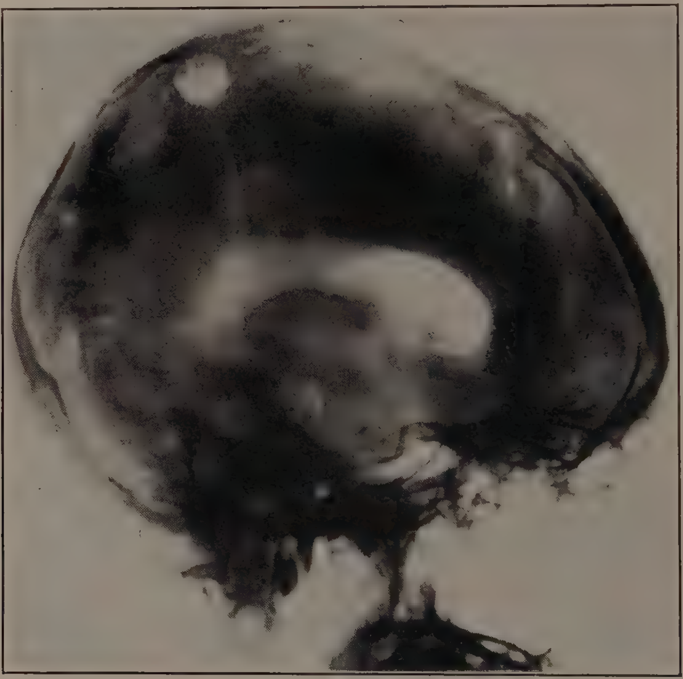

Fig. 260.—Normal lateral pneumoventriculogram. The light area is the air-filled ventricles.

Hysterosalpingography (Fig. 266) reveals the position, size, shape, contour, and patency of the cervical and uterine cavities, and the patency of the fallopian tubes.

Cholecystography (Fig. 267) gives information as to the size, shape, position, and relationship of the gall bladder, and in addition, demonstrates the ability of the organ to concentrate its contents, which is one of its certainly known functions.

A new substance, known as Uroselectan (Fig. 268), has been recently developed. This is administered by intravenous injection. It is almost wholly excreted in the urine, in which it reaches a concentration which renders the kidney, pelvis and ureters, and to a lesser extent, the bladder, opaque to the x-rays, and hence visible.

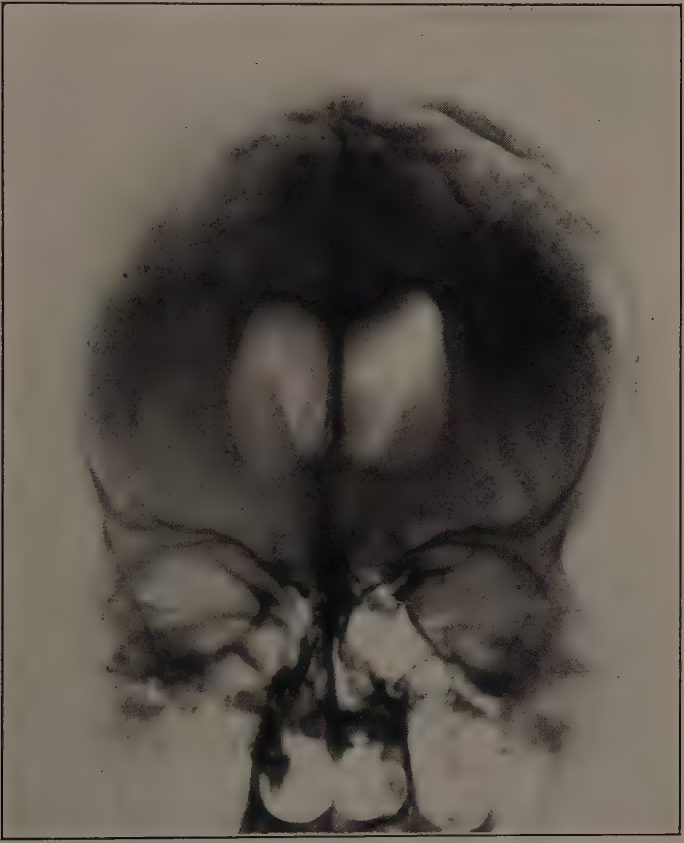

Fig. 261.—Postero-anterior view of Fig. 260.

The preceding pages are an attempt to elucidate the reason the phenomena of the x-ray can be usefully applied to the study of the human organism. Elaboration of this method of examination and its extension by artificial means has been discussed. Through-

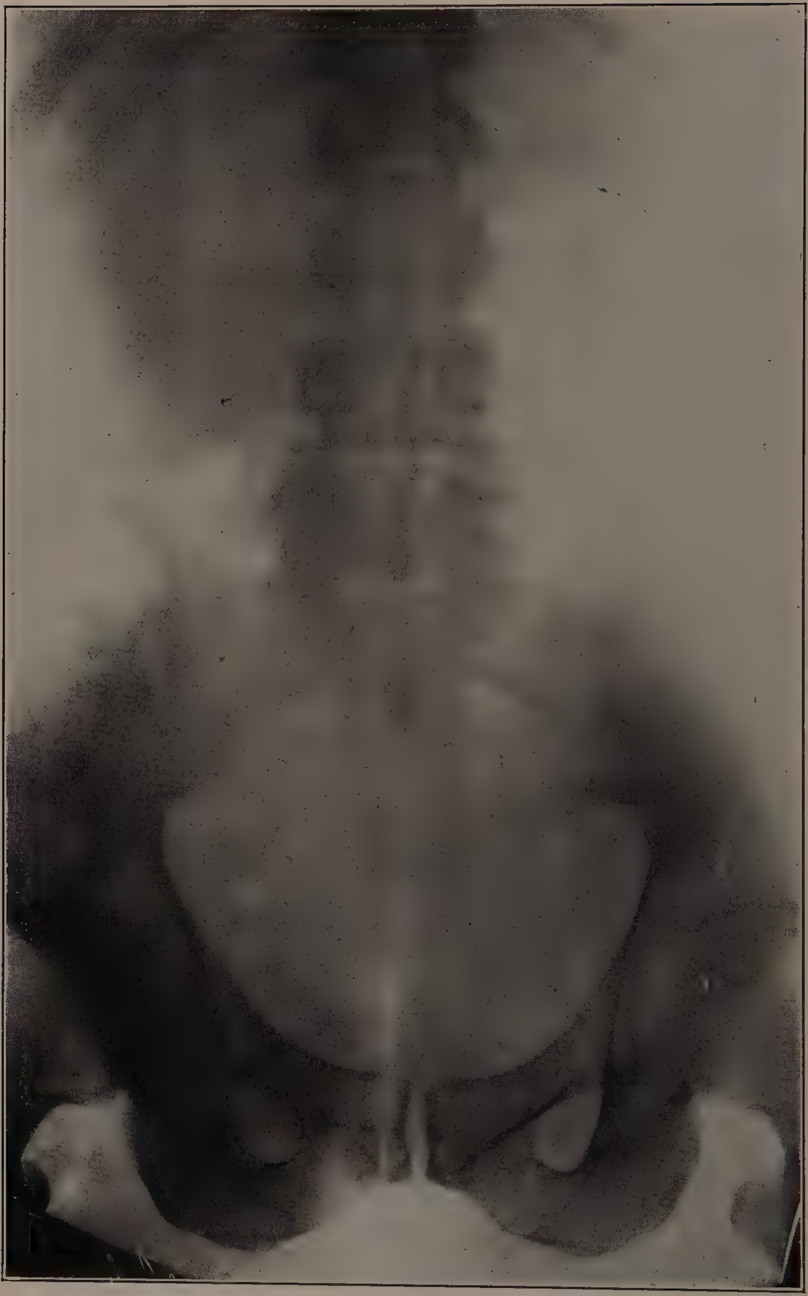

Fig. 262.—Radiograph of spine. Collection of iodized oil is seen at the lowest point of the spinal canal, it having been injected into the cysterna. Its descent to its present location proves the canal to be patent.

out, the endeavor has been to develop the principle that x-ray applications are based on physical phenomena entirely. The fact that the fluoroscopic or radiographic image in a sense constitutes a "picture" has resulted in the all too prevalent belief that x-ray studies result in pictures of this or that pathologic condition. In point of fact, these images are not pictures in the true sense of the word, but are only the record of the relative densities of the structures which lay in the path of the beam of the x-rays. It is the application of this principle which results in the radiographic or fluoroscopic images. These often enable the radiologist to make a final and complete diagnosis even when other methods of examination have failed; however, it must be remem-

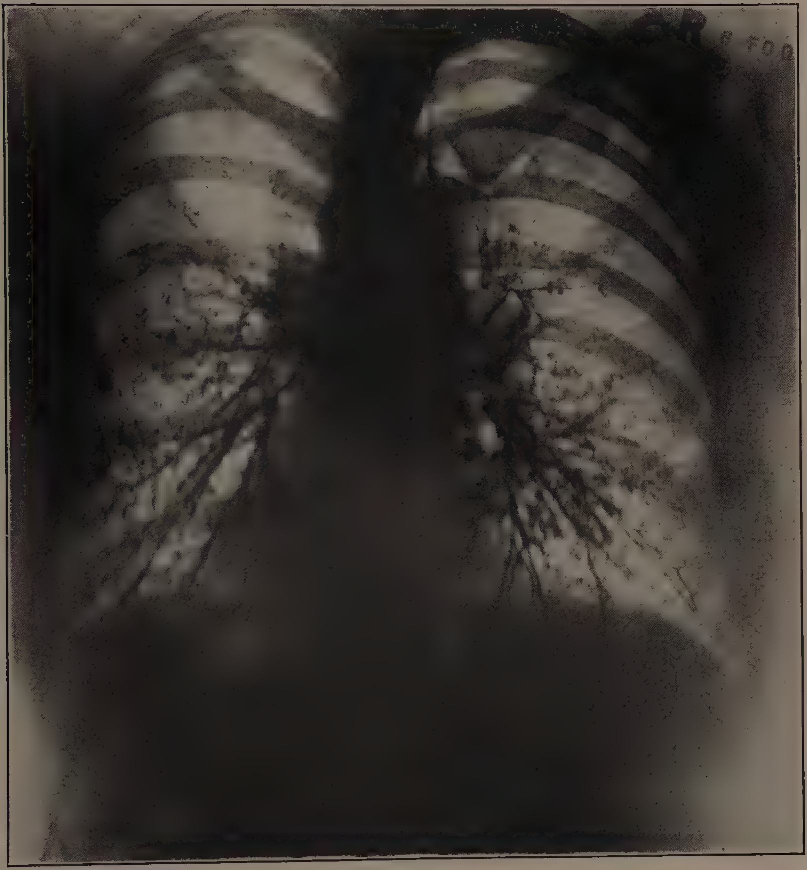

Fig. 263.—Bronchography. Note that the iodized oil reveals the course, caliber, and relation of the bronchial branches.

bered that radiologic examination should be used in conjunction with, but never to the exclusion of, other diagnostic measures.

Differences of density engendered by pathologic processes should be given attention at this juncture. They should be treated only in a general way and with the purpose of elucidating the underlying general principles involved. Radiologic consideration of pathologic processes as such will be found in the sections dealing with the diagnosis of specific disease states.

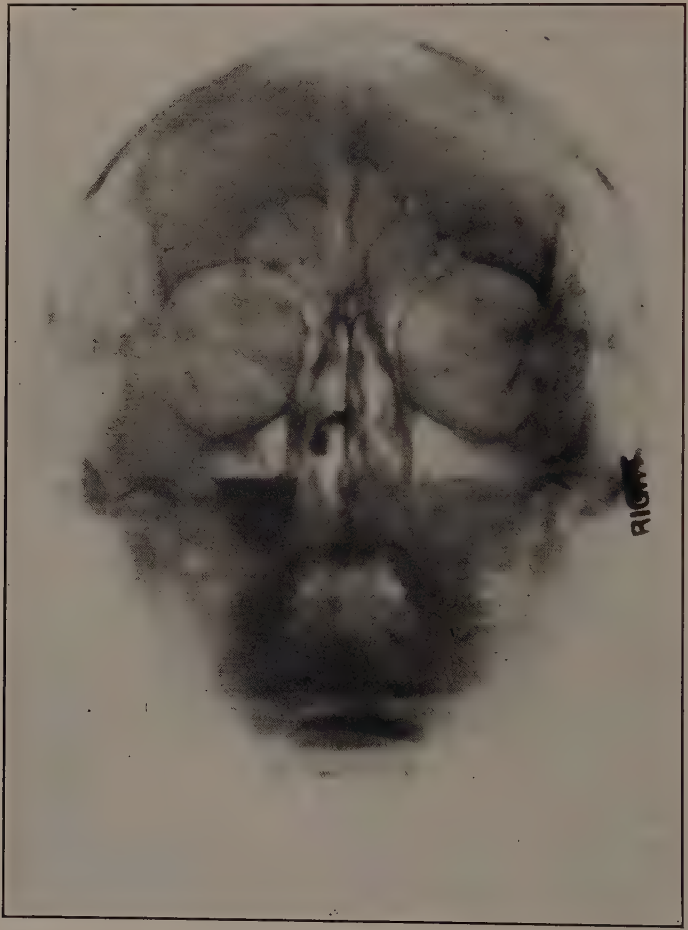

Fig. 264.—Postero-anterior view of maxillary sinuses with the iodized oil in the left maxillary antrum.

The first group of conditions to be considered comprises those with a density lying above the base line of uniformity. That is, those with calcium or other mineral content—the skeleton.

In addition to the differences which are physiologic, bone can become either more or less dense through pathologic processes. The effects of dietary insufficiency on the skeleton have been mentioned. This results in a decrease in the mineral content of the bone. Such changes due to diet are found most commonly in children, the two resultant diseases being rickets and scurvy. In adults, the condition known as osteomalacia is probably the counterpart of rickets in children. Discussion of these conditions has no place in a textbook on physical diagnosis.

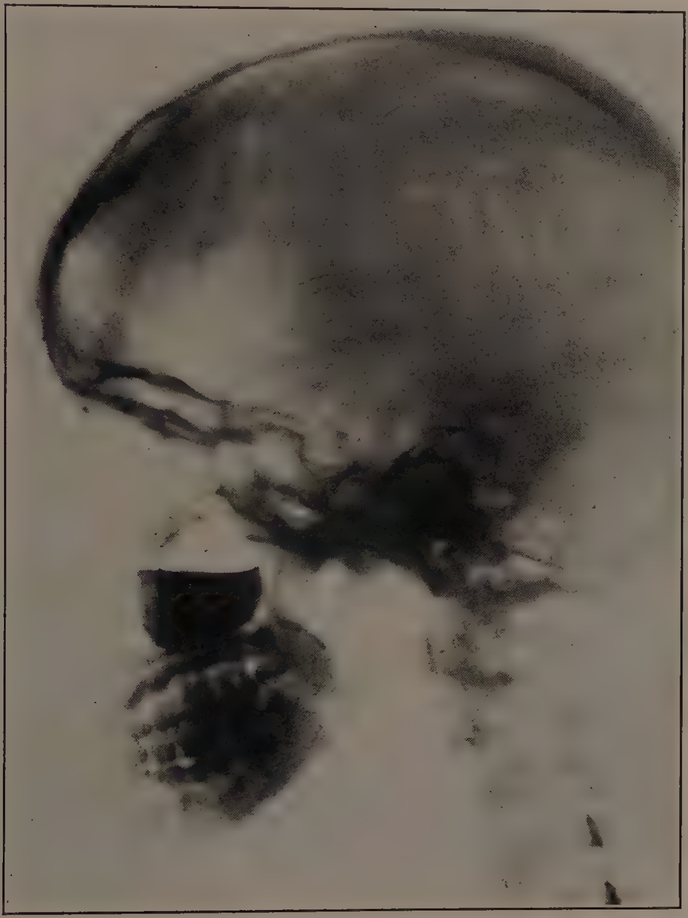
Fig. 265.—Lateral view of Fig. 264.

The conditions named above are of a type in which the amount of calcium throughout the skeleton is generally, not locally, diminished. Localized diminution of the mineral content of bone is encountered in skeletal tumors, both primary and secondary, trauma, and in bone infections.

There is an absolute increase in calcium content in certain pathologic conditions. This is usually termed sclerosis of the bone. It is a manifestation of a repair process, a common sequel to bone infections, arising most frequently from pyogenic and less often from syphilitic osteomyelitis.

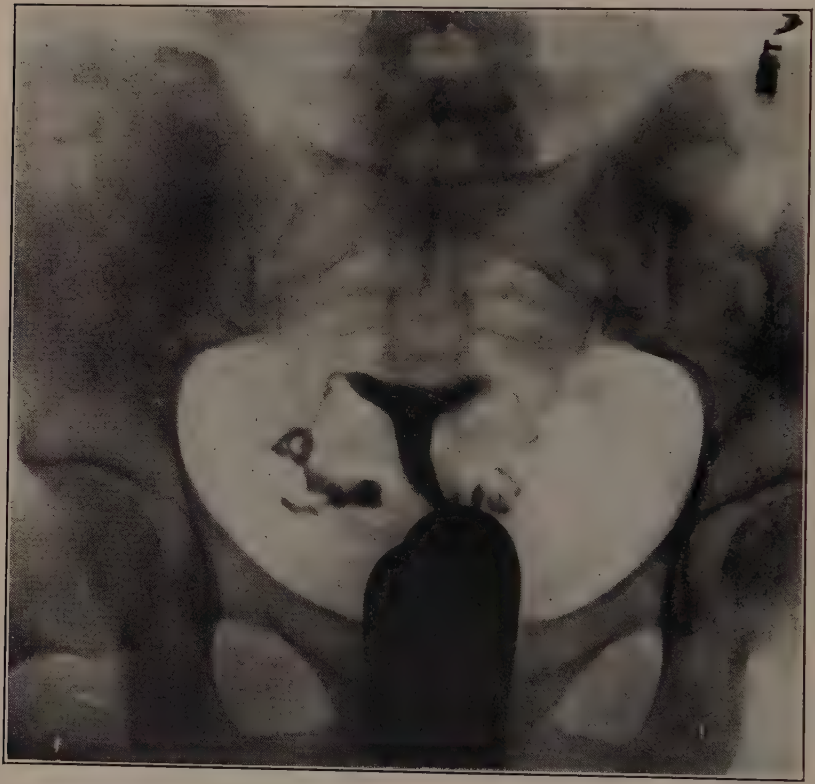

Fig. 266.—Injection of iodized oil into the uterine cavity and thence into the fallopian tubes.

Physiologic calcifications observed in the body have already been discussed, but the finding of calcified material where it is not normally encountered, comes in the realm of pathology, and merits special attention. Chief among the pathologic calcifications in point of frequency are those of healed inflammatory lesions, which are most commonly tubercular. Such calcifications are commonly situated in healed foci in the parenchyma or the hilus of the lungs. Calcified healed lesions are also, though infrequently, observed in the kidneys, liver, and spleen. They are of approximately the same causation as are calcified areas in the lungs.

Of much greater importance, though less frequently observed, are the calcifications forming calculi of various types, which are, in the order of the frequency of their occurrence, biliary, urinary, salivary, and prostatic calculi. More rarely, other calculi, such as those arising in the pancreas, are observed. Biliary (Fig. 269) and urinary calculi (Fig. 270) are of great clinical importance (salivary calculi are less so). However, it should be borne in mind that approximately 50 per cent of the biliary calculi do not contain enough calcium to differentiate them from the soft structures which surround them. Such gallstones are composed almost wholly of cholesterol which lies in close proximity to the base line of uniform body density. There are occasionally examples of urinary calculi, consisting chiefly of urates, to which the preceding statement applies.

Ossified tissues, hence those which contain calcium, are also occasionally observed in surroundings where the presence of calcium would not be anticipated. For example, osteogenetic tumors can produce ossifications in the soft structures at a distance from the parent bone. Violence may dislocate fragments of bone and deposit them far from their original position. There are cases of heterotopic bone formation where this tissue may be found in parenchymatous organs, muscles, etc. Such cases are, however, a great rarity.

Not only calcification, but, at times, also ossification, takes place in old hemorrhages, usually in the muscles or intermuscular septa. The most common occurrence of ossified or calcified hemorrhage is that which follows trauma to the muscles of the thigh, arm, or shoulder.

A final allusion to the teeth is in order. Teeth may be found occupying bizarre positions, in either jaw, and are not infrequently dislodged into the maxillary antra in the course of attempt at extraction. A curious phenomenon is that rudimentary teeth are sometimes observed in dermoid cysts.

In turning from a physiologic to a pathologic consideration of the structures containing substances whose density is below the general base line, it may be noted that the air content of the nasal passages and their accessory sinuses is profoundly affected

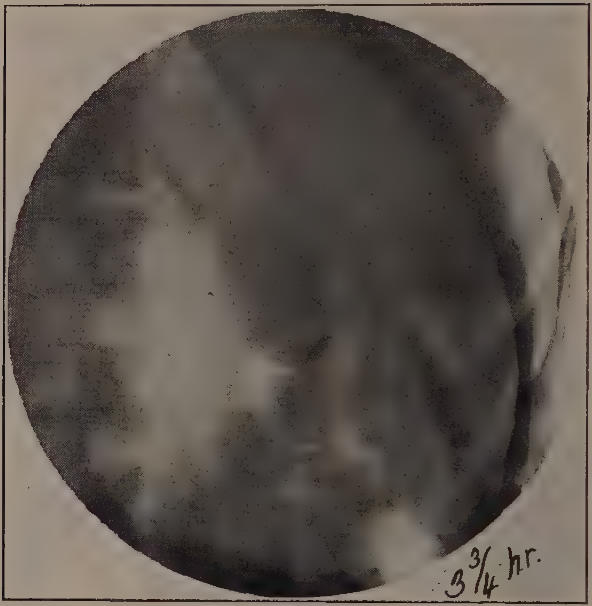

Fig. 267-*A*.

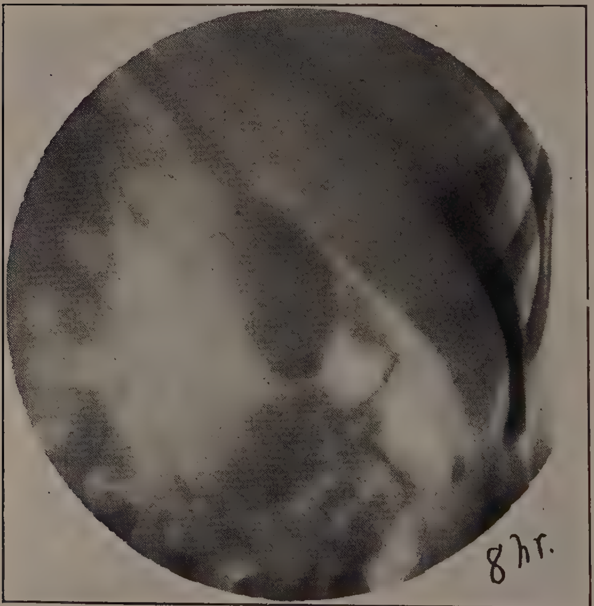

Fig. 267-*B*.

by disease. The air volume which the sinuses contain can be diminished by any condition which will increase the soft tissues normally contained within their bony walls, or the products of such tissues. This may vary from complete absence of air when the sinuses will appear homogeneous, down to such a slight diminution of air that it is scarcely perceptible. Observations in case of the nasal passages are similar, but the contrasts are far less marked. Pathologic conditions in the mastoid bones show, in addition to the alteration of the volume of air in the pneumatic structure, alterations in the bones themselves. Bone changes are but rarely noted in disease of the paranasal sinuses. The pneumatic structure of the mastoid may be totally devoid of air, and the bony septa separating the various cells undergo solution. The air content of the mastoid, may, in pathologic conditions, vary from a complete absence of air down to a barely perceptible shading, due to a slight thickening of the linings of the cells.

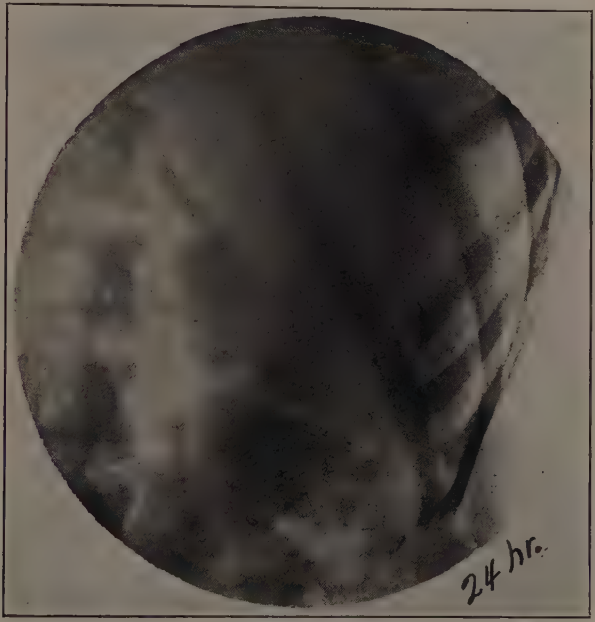

Fig. 267-*C*.

Fig. 267.—Normal cholecystograms showing the changes in the gall bladder at different hours of radiographic examination.

In pathologic conditions, the air column in the upper respiratory tract from the pharynx to the larynx will show intrusion of soft tissue structure only. Below the larynx, displacement of the air column of the trachea is a fairly common pathologic observation. Infrequently there is a slight degree of compression of the trachea. This is demonstrable only when it is lateral.

Under pathologic conditions, the air content in the lungs can be varied by either pleural or pulmonary processes. It may be added that an apparent increase of this density can be found in certain conditions which affect the thoracic walls, such as extensive edema. In such cases, and in fact, in all cases in which ap-

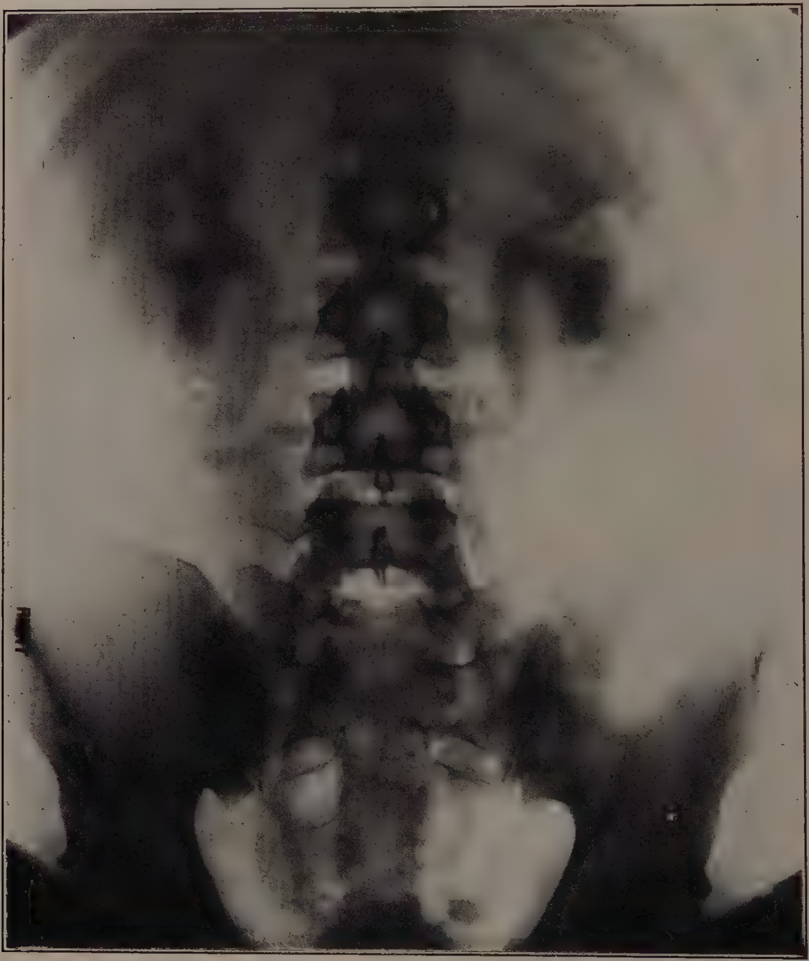

Fig. 268.—Pyelogram-uterogram-cystogram obtained after intravenous injection of Uroselectan. This is a pathologic case of dilated ureters and kidney pelves resulting from contracted bladder clearly seen at the bottom of the film.

parent decrease in aeration of the lungs is observed, this can be ascribed to a decrease in the volume of air relative to the total thoracic thickness. Any of the inflammatory processes which result in the filling of the alveolar air spaces with something other than their normal air content, are those which increase the density of the lung.

A similar physical change results from the absorption of alveolar air in the presence of an obstruction which prevents the ingress of additional air (the so-called obstructive atelectases). Either the whole of, or a large part of, the lung may show a decreased volume of air. Complete consolidation of the lung, or of a single lobe, can be observed in lobar pneumonia. There are processes which produce discrete areas devoid of aeration. These areas vary in size according to the type and intensity of the pathologic process.

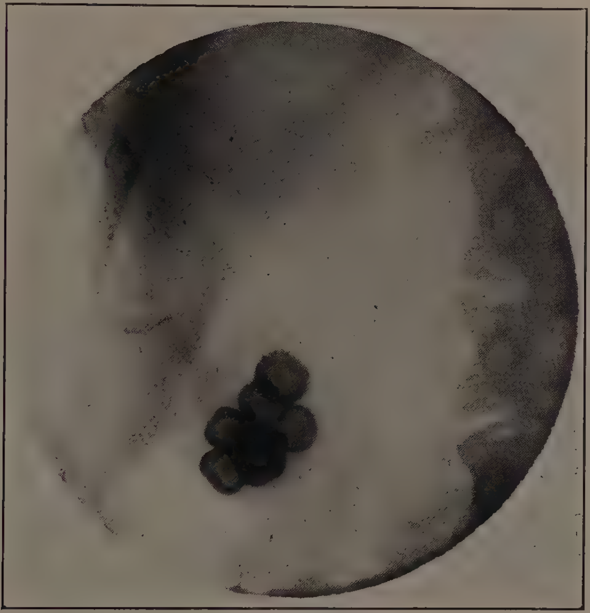

Fig. 269.—Multiple calcified biliary calculi.

A focus in the lung may so disintegrate through a pathologic process that a cavity is formed. This may or may not be filled with fluid material. In the event that the contents are fluid, such a cavity appears as an area of density which is indistinguishable from that of an infiltrating process. If a cavity drains itself of its fluid contents so that it contains air, then such a density contrast is found as obtains when air is present in tissues and structures where it is not normally present. That is true even to the extent that a pulmonary cavity containing air is less dense than is the surrounding aerated lung. Pulmonary cavities, however, except when located toward the apex of the lung, almost always appear as areas of increased density either because of their

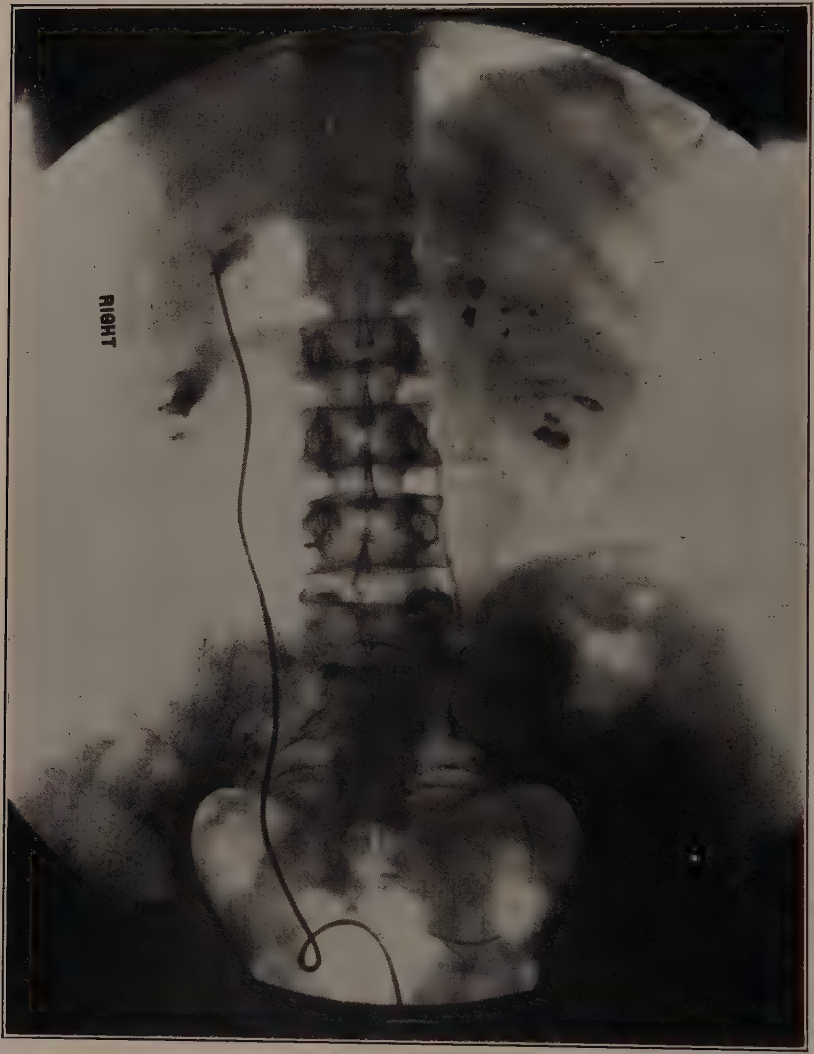

Fig. 270.—Multiple bilateral calcified urinary calculi. Opaque catheters are in both ureters.

thick walls or because of their fluid contents. These important facts should be carefully noted.

A pathologic condition involving the pleura and causing it to thicken or to produce fluids probably always affects the air content of the lung. From the standpoint of x-ray procedures, the student should think of fluid collections in the pleura as being *fluid,* and should not consider them as being blood, exudate, transudate, or pus. All such fluids approximate each other so closely in specific gravity that there is no possibility of distinguishing between them directly by means of x-ray examination. The same is true of the consolidated lung of any causation, and solid pleural tumors. While points of differentiation between these conditions do exist, they are based on secondary factors, such as the dislocation of the heart to the side opposite a pleurisy with effusion, and its dislocation toward the opaque side in cases of massive collapse of the lung. There is nothing intrinsic in the appearance of an opaque lung observed by x-ray which enables one to specifically diagnose any one pathologic process.

In this connection it may be said that herniations of the abdominal contents into the thoracic cavity can be discovered both when they are solid and when they contain some portion of the alimentary tube containing gas.

The aerated lung outlines the contour of the heart, thereby revealing enlargement, hypertrophy, and alteration of various chambers of the organ. Specific valvular lesions are sometimes thus discovered. The heart rate and the heart excursion can be observed and noted, as can its mobility or fixation. Dislocation of the heart and situs inversus are readily demonstrated. Dilatation and aneurysm of the first portion and arch of the aorta are well shown. Saccular aneurysms in the anterior and posterior aspects of the aortic arch and aneurysm of the descending portion of the aorta down to the diaphragm are less readily recognized. Beyond that point, the homogeneity of the abdominal structures prevents any differentiation at all. Erosion of the bony elements of the thoracic cage by aneurysm does not depend upon the air content of the lungs for demonstration.

It has been said that edema of the thoracic wall produces a haziness over the lung suggesting a decrease in air content. It should be noted that the breasts in women always cause a similar slight haziness over the lower portion of both lungs. If there has been an amputation of the breast, there will appear to be a difference in density in the two lungs because of the lesser volume of the soft tissue on the side on which the amputation has been performed. This is another proof, if any proof be needed, that radiologic procedure is a method of indirect visualization, and because it is such, the axiom of comparing one side of the body with the other should always be carried out. This principle cannot be too frequently stressed.

Mention has already been made of the x-ray image produced by cavities of the lungs. From the standpoint of radiologic examination, dilatations of the bronchial branches, because of their thick walls and fluid contents, should be placed in the same category. It is in such cases that bronchography, in which procedure iodized oil is injected, is employed. This reveals the size, shape, position, capacity, and relationship of sacculation or fusiform dilatations of the bronchial branches. Abscess cavities can be discovered by bronchography, whereas with the ordinary radiograph of the chest they appear only as infiltrations.

The references which have already been made to the presence of gas in the tissues, as in emphysema, pneumatocele, pneumothorax, should be considered in more detail. Rupture of the walls of the upper respiratory tract which connect with one of the adjacent cavities may result in the collection of air where it is not customarily present. Fracture of the base of the skull may result in the formation of a pneumatocele within the cranial cavity. Subcutaneous emphysema arising from trauma may involve the cellular structure of the head and neck, and at times this spreads over the entire body. Rupture at a lower level can result in the collection of air in the mediastinum, and even in the pericardial sac. The most common occurrence of this sort in the respiratory tract is the appearance of pneumothorax, the collection of air in the pleural cavity. Not only the presence of an opening connecting any of the cavities mentioned to the main air passages, but also the respiratory act itself is an important factor in the production of pneumatocele, subcutaneous emphysema, or pneumothorax. It would appear that something in the nature of a valve action at the point of rupture is necessary for the production of any of these conditions.

In this connection it may be said that air is introduced into the tissues in the course of surgical operation. A small amount of air remains in the abdominal cavity following many laparotomies. It remains, however, for only a short period of time, and is of no consequence. Air or gas in the peritoneal cavity follows upon the perforation of the hollow viscera therein contained and produces a spontaneous or pathologic pneumoperitoneum. Gas-producing organisms can result in the collection of gas in almost any portion of the body.

As a rule, solid foreign bodies are visualized when present within the body, because of their greater density; however, solid foreign bodies of a lesser density than their surrounding soft tissues can be observed at times; for example, cholesterol gallstones are thus occasionally observed. But the most notable example is that of foreign bodies of lesser density than that of the general base line which can be observed in the lower respiratory tract because they are in contrast to air-containing tissue. This does not occur as commonly as one might suppose. In order that such a thing can happen it is necessary that the foreign body be located well out in the parenchyma of the lung. Since the bronchial passages rapidly decrease in size from the root to the periphery of the lung, only small foreign bodies can reach a distance sufficiently far out for them to be surrounded by the aerated lung tissue which would serve to differentiate them. When they are too small they will escape observation in any case; however, such foreign bodies are occasionally seen.

It is extremely important that the student divest himself of the notion that x-ray study presents "pictures" of one sort or another. It should be reiterated that it reveals only the variations in the rate of absorption of x-rays in the tissues and structures interposed in the path of the beam of x-rays. When a variation from the normal rate of absorption of the x-rays is noted, explanation should be sought for the causes of such a phenomenon. The student should inquire, "What is the pathologic process which has altered the density of the examined structures and resulted in a difference of x-ray absorption?" When such an attitude has been assumed, and when his mental approach to diagnostic radiology is in the light of a physical measurement, the student will have overcome the main barrier which lies in the way of an intelligent conception of the function and application of radiology in physical diagnosis.

CHAPTER XXXIV

ELECTROCARDIOGRAPHY

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Assistant Professor of Clinical Medicine, Washington University School of Medicine

Electrocardiography has developed to such proportions that a comprehensive treatise on the subject could not find place within a single volume devoted to physical diagnosis. The aid which it gives in the diagnosis of certain diseases of the heart and disorders of the heart beat has become so important, however, that such a book would be lacking in completeness if it did not include at least a brief account of the elements of this important subject. Our plan is to present the principle of the electrocardiograph and to describe the normal electrocardiogram, along with a brief discussion of its component parts. As the various cardiac abnormalities are successively discussed in the second part of the text, reference will be made to the accompanying electrocardiogram in those instances in which the latter is of diagnostic value. A few characteristic records will be shown. No attempt has been made to make the list of these records complete or to make the discussion of them comprehensive. The purpose has been rather to limit both, so as to keep them within proper compass. With such a basis the student may refer to special works on electrocardiography as occasion arises.

PRINCIPLE OF THE ELECTROCARDIOGRAPH

Muscular activity is accompanied by change in electro-potential. The electrocardiograph is an instrument devised to record such changes as they are developed in the heart. The instrument consists of a galvanometer and certain accessory parts so arranged as to produce a record of these changes in potential. By means of electrodes and small wires the cardiac current is led away and carried through the machine; the heart, the electrodes, the wires and the galvanometer thus constituting an electric circuit. In experimental work the electrodes may be attached directly to the heart. In human subjects such *direct* leads, of course, cannot be used, but the electrodes are attached to external parts which, through their anatomic connections with the heart, constitute *indirect* leads. Any two convenient locations might be used as such "leads." But it has been found that most satisfactory results are given by employing three standard leads; viz.: (a) Right Arm to Left Arm, which is designated as Lead I; (b) Right Arm to Left Leg, Lead II; (c) Left Arm to Left Leg, Lead III. An electrocardiogram may be made from any one of these leads, but certain information is obtained from using all three which is not available from one lead alone, in much the same way as photographs made from various angles give more information than does a single picture presenting only one view. In the routine taking of an electrocardiogram, therefore, three records are made, one each, from Leads I, II, and III.

There are two types of machines in general use. In one the cardiac current is led through a circuit containing a string galvanometer, each change in electro-potential producing certain characteristic movements of the string. By projecting the shadow of the string upon a moving photographic film, a photograph of the movements of the shadow is obtained. This photographic record is an electrocardiogram. The machine is equipped with a time marker, the completed record showing in graphic form certain events of the cardiac cycle with the time relationship of their occurrence.

In instruments of the second type, the cardiac current is amplified and led through a circuit embracing a mirror galvanometer, the changes in electro-potential producing oscillations of the mirror. A beam of light plays on the mirror, and as the latter oscillates the reflected beam is projected on a moving photographic film. Electrocardiograms produced by instruments of one type are quite similar to those produced by the other. For detailed description of the instruments the student is referred to textbooks of physics and to special works on electrocardiography.

THE NORMAL ELECTROCARDIOGRAM

Curves from normal subjects show minor differences and no two are exactly alike, but the general form of normal human electrocardiograms is constant and shows certain characteristic waves. These are usually lettered after the terminology of Einthoven which is employed in the accompanying figures.

In Fig. 271 the three leads of a normal electrocardiogram are shown. Two broad waves lettered *P* and *T* and a sharp deflection lettered *R* occur with each cardiac cycle in the record from each lead. In the second and third leads of Fig. 271 there is a very small downward deflection lettered *Q* just before the *R* wave. This wave is absent or quite small in the majority of electrocardiograms, but it is by no means uncommon. (See Figs. 272, 321.) Figs. 272 and 273 show other examples of normal electrocordiograms. In

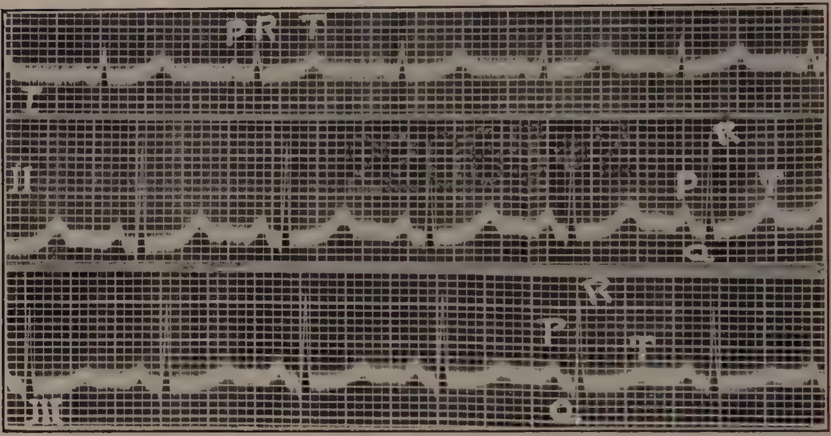

Fig. 271.—Normal electrocardiogram, Leads I, II, and III. The time interval between two of the heavier perpendicular lines is one-fifth of a second.

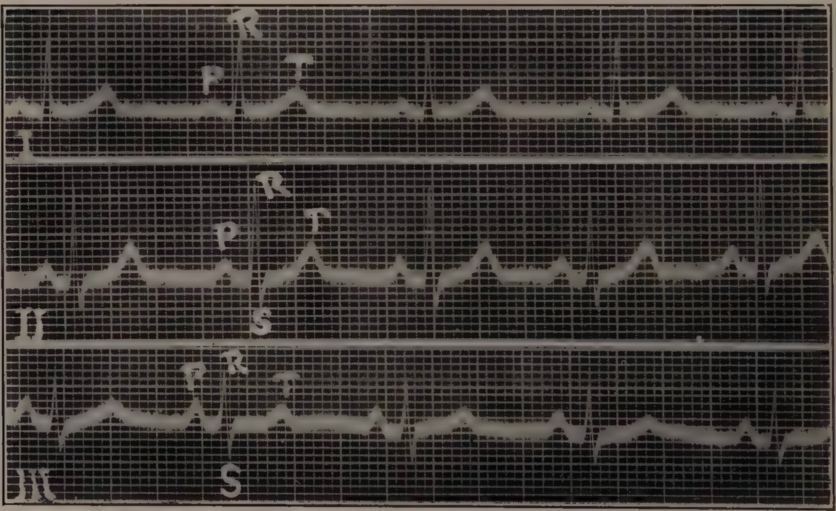

Fig. 272.—Normal electrocardiogram, Leads I, II and III.

Leads II and III of Fig. 272, and in Leads I and II of the first and second records of Fig. 273 there is a downwardly directed wave lettered *S* immediately following the *R* wave. The *S* wave in some records is of considerable amplitude. This will be discussed later in connection with consideration of the electrical axis. Here it may be said that in typically normal records the amplitude of the *S* wave is less than that of the *R* wave.

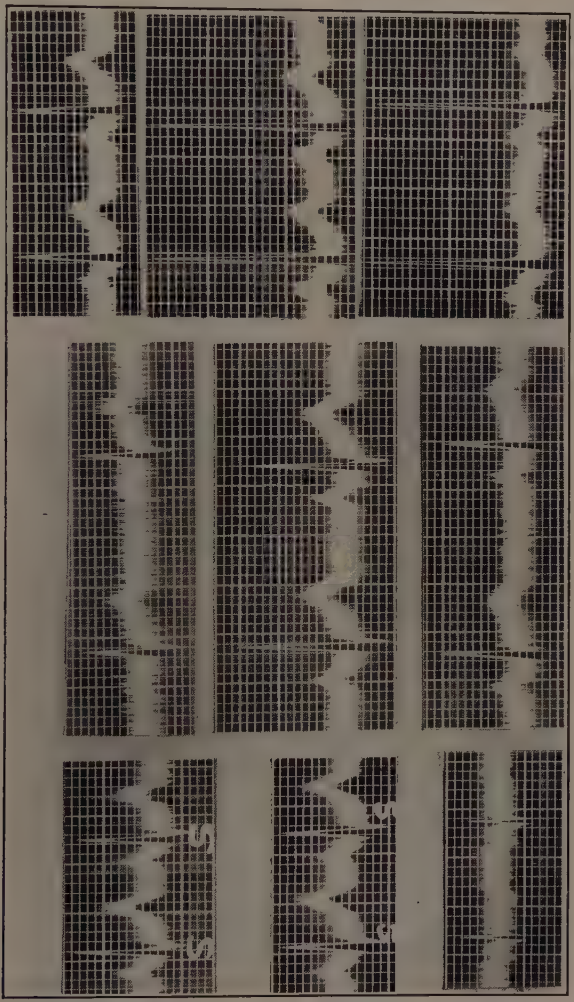

Fig. 273.—Three specimens of normal electrocardiograms, Leads I, II and III.

Before discussing the separate components of the normal electrocardiogram, it will be helpful to review briefly the mechanism of the normal heart beat. That property of heart muscle by virtue of which it initiates spontaneous contractions is called rhythmicity or automaticity. There is much evidence that it resides only in the specialized tissues. Automaticity is highest at the *sino-auricular* node and this area, therefore, becomes the pacemaker. The impulse to contraction, arising at the pacemaker, spreads over the auricle and reaches the *auriculo-ventricular node.* This is a mass of specialized tissue which lies in the lower part of the auricular muscle at the base (posterior right border) of the auricular septum. The node constitutes an enlargement near the beginning of the *auriculo-ventricular bundle,* a specialized strand which runs downward to the membranous part of the ventricular septum. Here it divides into a right and left branch, one upon each side of the septum. Subdivisions of the branches are continued in the endocardium as the Purkinje cells, thus bringing the auriculo-ventricular bundle into contact with the musculature of the ventricle.

After the impulse has initiated the orderly contraction of the auricle, it is conducted to the musculature of the ventricle along this path. It reaches the various parts of both ventricles at about the same time and causes a contraction of these chambers which is almost simultaneous in all parts of both. This sequence of events is portrayed in the electrocardiogram. Experimental evidence proves that the *P* wave is produced by auricular activity and that the *Q R S T* group is due to activity in the ventricles. Between the *P* wave and the onset of ventricular activity there is an iso-electric interval which represents approximately the passage of the impulse from the auricle along the bundle and its branches. It is not known exactly at what stage in auricular activity the impulse enters the a-v bundle, however, and so it is customary to measure the "conduction time," not from the end of the *P* wave, but from its *beginning,* for the upstroke of the *P* wave is known to be inscribed at or just after the beginning of activity at the sinus. The conduction time, or the *P-R* time as it is often called, may be defined, then, as the time (measured in hundredths of a second) between the beginning of the *P* wave and the beginning of the *Q* or *R* wave.

The P-R time differs in different individuals. The standard of measurement is that of the second lead. The normal limits are given by Lewis as from 0.13 of a second to 0.21 of a second. Intervals less than 0.13 of a second are occasionally seen, especially in young children. Intervals of more than 0.18 of a second create the suspicion of impairment of conductivity. In an electrocardiogram, then, is pictured with each systole, a graphic expression of the beginning of auricular activity at the pacemaker, its spread over the auricle, its transmission through the a-v bundle and its branches, and the muscular activity of the ventricles.

The electrocardiogram thus furnishes the most exact means at our disposal of diagnosing the *cardiac mechanism*. Reference to this use will later be made in the chapter on *Abnormalities of the Heart Beat*. Its use in another direction, however, becomes increasingly valuable. Since the ventricular complex (*Q R S T* group) constitutes a record of the variations in electro-potential developed in the muscle of the ventricle, a study of this ventricular complex often affords information relating to the ventricular musculature that can be obtained in no other way. In evaluating such a record, it is necessary to remember that each point of the ventricular complex represents the algebraic sum of all variations in potential developed throughout the muscle of both ventricles at the moment. Some of these effects tend to move the string or the mirror in one direction, some in another. Activity at one place, therefore, may tend to neutralize or to augment activity synchronously developed at another place. The normal ventricular complex is indeed a bicardiogram representing the effects produced synchronously in the right and left ventricles. The effect of activity in either ventricle alone would produce a record quite different from that produced by activity in the other ventricle alone. Such a record, obtained experimentally, of activity in the right ventricle alone, has been called a dextrocardiogram. It shows a deep *S* wave in Lead I and a high *R* wave in Lead III. An experimental record of the left ventricle shows a high *R* wave in Lead I and a deep *S* wave in Lead III. It has been called a levocardiogram. By superimposing the one upon the other and charting the resultant curve, a record which approximates the normal is produced. In order to appreciate variations in the normal ventricular complex, as well as certain abnormalities, this conception of the bicardiogram should be kept in mind. In certain records it will be discovered that their shape resembles one or the other of the component parts. In order to appreciate the significance of this resemblance, a conception of the electrical axis is necessary.

The Electrical Axis.—As the excitation wave moves over the ventricles, electromotive force is developed along the path travelled by the wave. The line along which the greatest electromotive force is being developed at any moment constitutes the electrical axis at that moment. Many parts of muscle in both ventricles are activated at the same instant, and after these, other parts are affected. The electrical axis, therefore, is constantly changing its direction. Its general course over the ventricle, however, describes a line which represents, broadly speaking, the direction in which the muscle fibers are successively activated. Now the direction of the movement assumed by the string or the mirror of the electrocardiograph, and, therefore, the direction of the resulting wave in the electrocardiogram, depends on the relationship of the electrical axis to the axis of the lead employed in taking the record. By taking all three leads and comparing the amplitude and direction of the *Q R S* complexes in them, the electrical axis may be computed.

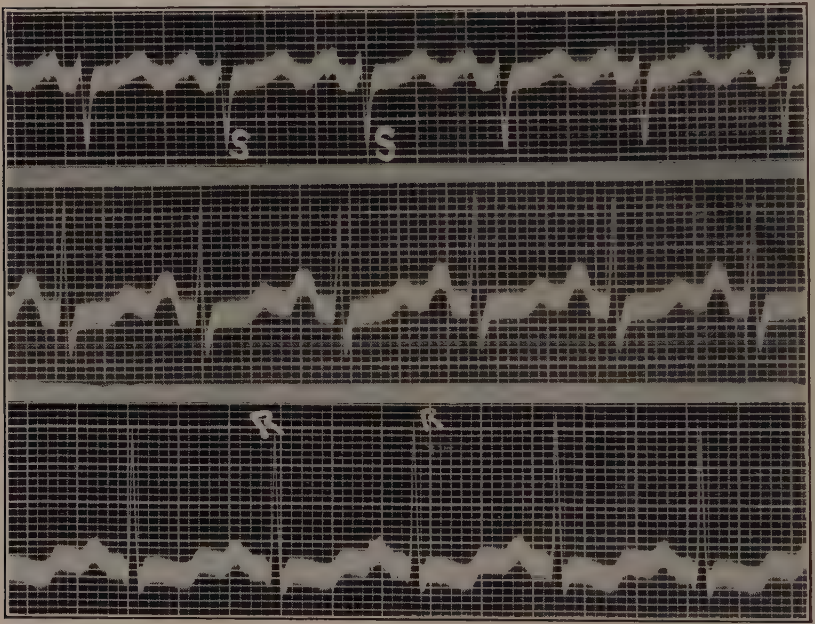

Fig. 274.—Right axis deviation. Discussion in the text.

In a general way the direction of the electrical axis corresponds roughly to the anatomic axis or the "cardiac axis." Whatever causes the heart to change its position in the chest, therefore, may be expected to produce a similar change in the electrical axis, and, consequently, in the *Q R S* complexes in the three leads of the electrocardiogram. Certain factors other than position of the heart, however, are common causes of an atypical or abnormal

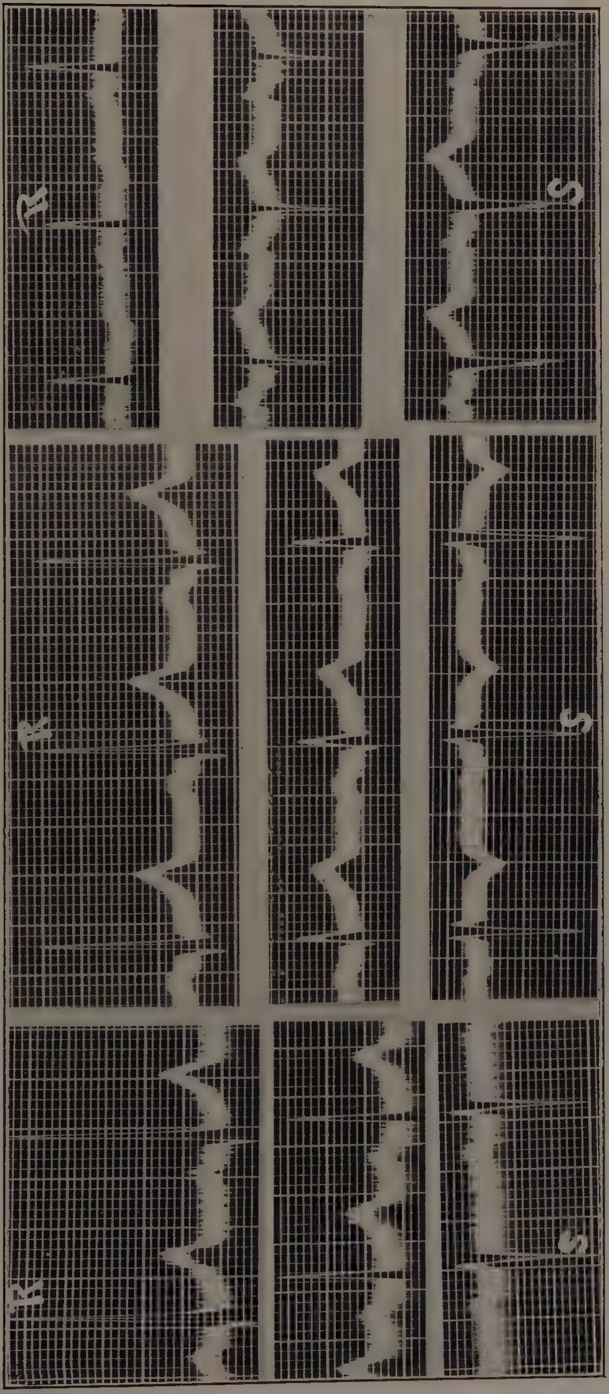

Fig. 275.—Specimens of three electrocardiograms showing left axis deviation. In each record the *R* wave predominates in Lead I and the *S* wave in Lead III, no matter what may be the direction of other waves.

electrical axis as depicted in the electrocardiogram. (a) Difference in conductivity between the two branches of the a-v bundle may result in the excitation wave reaching one ventricle before the other, thus causing the electrical axis to deviate toward the side first activated and producing a record resembling a dextrocardiogram or a levocardiogram, as the case may be. Thus right axis deviation may result from defective conductivity in the left branch of the bundle, and left axis deviation may be due to impairment of conductivity in the right branch. (b) It is commonly believed that disproportion between the masses of muscle in the two ventricles influences the direction of the electrical axis; relative hypertrophy of the left ventricle producing left axis deviation and vice versa. Fig. 274 is an example of right axis deviation. Note the deep *S* wave in Lead I and the tall *R* wave in Lead III. Fig. 275 shows three examples of left axis deviation. The *R* wave is tall in Lead I and the *S* wave deep in Lead III.

Since the effect of such factors, strictly speaking, is to produce abnormalities of the electrocardiogram, discussion of them might more properly find place in the second part of the book. Mention of them is made here, however, because an understanding of them is helpful toward an appreciation of the normal electrocardiogram and of the factors responsible for the form of its ventricular complex. It will be observed that the ventricular complex is composed of two parts: (a) an initial deflection, the *Q R S* group, and (b) a final deflection, the *T* wave. The initial deflection (*Q R S*) results from activity first developed in the ventricles as the impulse from the auricle reaches the ventricular muscle after its passage along the a-v bundle and its branches; the amplitude and direction of the initial deflection being influenced by the factors above noted. The exact mechanism responsible for the production of the *T* wave, and therefore, for its direction in any lead, is less well understood. A review of theories here would be out of place. It may be stated, however, that an inverted *T* wave in Lead I rarely if ever can be considered normal. In Lead III an inverted *T* wave cannot, *per se,* be regarded as abnormal. The height and direction of the *T* wave in Lead II (and also of other waves in this lead) are approximately the algebraic sum of these factors of the wave in Leads I and III. In normal records the *T* wave should not be inverted in the second lead.

PART II

PHYSICAL DIAGNOSIS OF DISEASES OF THE RESPIRATORY AND CIRCULATORY SYSTEMS

SECTION I

DISEASES OF THE RESPIRATORY SYSTEM

CHAPTER XXXV

DISEASES OF THE TRACHEA

TRACHEITIS

Acute Tracheitis.—*Clinical Pathology.*—Acute tracheitis is an acute inflammation of the mucous membrane of the trachea occurring usually as a complication of acute inflammation of the nasopharynx and extending to the primary bronchi. The disease may be a part of an acute coryza or may be the result of inhaling irritating fumes, such as bromid iodine, the irritating gases used in war, or "tear bombs."

During the early stages the mucous membrane is swollen, red, and dry. Later, it may be covered with a mucus or purulent exudate.

The physical signs on inspection are usually negative, except for slightly restrained respiratory movements. Palpation is negative, as is also percussion. Auscultation usually reveals harsh breath sounds over the upper sternum, occasionally accompanied by large moist râles.

The diagnosis depends largely on a harsh cough, pain over the upper sternum, and harsh breath sounds with occasional large moist râles.

Chronic Tracheitis.—Chronic tracheitis is usually found in excessive users of tobacco or individuals exposed to irritating gases. The physical signs resemble those of acute tracheitis.

TRACHEAL STENOSIS

Stenosis of the trachea may result from acute inflammatory disease, such as diphtheria, inhalation of irritating gases, or edema due to allergy.

Foreign bodies may pass the larynx and lodge in the trachea, causing partial occlusion.

Tumors within the trachea or tumors of the thyroid gland or mediastinum, producing pressure from without, cause gradually progressing stenosis.

The physical signs on inspection are cyanosis, depending on the degree of stenosis, inspiratory retraction of the lower intercostal spaces and epigastrium. Dyspnea in severe cases may be both inspiratory and expiratory.

Palpation may reveal a distinct palpable "wheeze" as the air passes over the obstruction. Percussion shows nothing, unless a tumor, such as an aneurysm, may be mapped out.

Auscultation may reveal harsh crowing inspiration, more marked over the manubrium. The diagnosis rests on the history and the localization of physical signs over the manubrium.

Bronchoscopy may reveal a foreign body, swelling and edema of the affected area, or the evidence of external pressure.

X-ray examination may localize a tumor or foreign body. A narrowing of the tracheal lumen or displacement of the trachea may also be revealed.

Differential Diagnosis.—*Laryngeal Stenosis.*—In laryngeal stenosis there is a vigorous respiratory movement of the larynx, the head is fixed and thrown somewhat backward, and respiration is stridulous.

The laryngoscope reveals the cause of the stenosis.

Tracheal stenosis is attended with less stridor, with limitation of the laryngeal movement during respiration, and frequently the patient is orthopneic.

Fluoroscopy or bronchoscopy may reveal the cause of the stenosis.

CHAPTER XXXVI

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

Clinical Pathology.—Acute bronchitis, an acute catarrhal inflammation of the mucous membrane of the medium-sized and larger bronchi, occurs as a primary affection, and as a complication of many of the acute infectious diseases, notably the exanthematous fevers, influenza, typhoid fever, and malaria.

The disease is most prevalent during the sudden changes of early spring and late autumn. Among predisposing causes may be mentioned particularly acute coryza, affecting the upper respiratory passages; and passive congestion of the lungs incident to regurgitant heart disease, acting upon the lower portions of the bronchial tree. The organism which is most frequently causative is the pneumococcus, alone or in conjunction with the staphylococcus, the colon bacillus, the micrococcus catarrhalis, or the bacillus typhosus. Irritating gases may also be a causative factor.

During the early stages of the inflammation the mucous membrane of the bronchi is swollen and red, but is dry. During the further evolution of the disease, however, the congested mucous membrane becomes bathed with secretion, mucopurulent or purulent in character, containing large numbers of desquamated epithelial cells and bacteria.

Physical Signs.—Mild cases of acute bronchitis yield few physical signs which are characteristic of the affection. In the more severe grades of bronchial inflammation physical signs are more in evidence, but often require a very careful examination to afford definite diagnostic data.

Inspection in the milder cases usually reveals nothing abnormal, but in more severe cases there is moderate acceleration of the respirations; while if there is a complicating or concomitant inflammation of the finer bronchioles (capillary bronchitis), the condition is attended by a considerable degree of dyspnea, and in the young subject, cyanosis.

Palpation, negative in cases of moderate severity, in well-developed cases of acute bronchitis may reveal slight rhonchal fremitus distributed over both lungs.

Percussion seldom elicits any alteration of the normal vesicular resonance in cases of frank acute bronchitis. Occasionally in very severe cases a slight impairment of resonance is evident over the bases posteriorly. The thorax should be carefully percussed daily in order that a complicating bronchopneumonia may be recognized in its inception.

Auscultation during the early stage of the disease reveals the presence of sibilant and sonorous râles well distributed over both lungs. In a later stage of the affection, after the secretion has become freely established, moist râles appear, the crepitant râle predominating the clinical picture.

Vocal resonance is not perceptibly altered. The respiratory murmur is harsh or puerile; but in uncomplicated acute bronchitis the breath sounds are never purely bronchial.

Diagnosis.—The diagnosis of acute bronchitis rests upon the absence of physical signs other than puerile breathing and a few râles distributed over both sides of the thorax, coupled with certain subjective symptoms, as an initial chill or chilliness followed by moderate fever, a dry hacking cough which loosens with the establishment of the bronchial secretions; a feeling of rawness and pain beneath the sternum; and a general feeling of malaise and pain in the back and limbs.

Differential Diagnosis.—In its abrupt onset the disease frequently is suggestive of *lobar pneumonia;* but this disease is eliminated by the absence of physical signs of consolidation; namely, increased vocal fremitus and resonance, flatness, and blowing, tubular breath sounds. Moreover, lobar pneumonia is almost invariably a unilateral affection, whereas acute bronchitis is bilateral in its manifestations. The constitutional toxemia and depression of lobar pneumonia far surpass that which attends acute bronchitis.

Bronchopneumonia is usually gradual and insidious in its primary manifestations; and, in addition to the physical signs of a diffuse acute bronchitis, may present multiple areas of impaired resonance, over which the respiratory sounds are bronchial or at the least are bronchovesicular. The constitutional disturbance accompanying bronchopneumonia is more pronounced, and the dyspnea is of a more extreme grade.

Pertussis, during the first week or ten days, cannot be differentiated from acute bronchitis; but after the development of the first ''whoop'' the diagnosis is readily made. A history of exposture to pertussis is often to be elicited. Moreover, pertussis is commonly attended by a striking lymphocytosis.

CHRONIC BRONCHITIS

Clinical Pathology.—Chronic catarrhal inflammation of the bronchial mucous membrane occurs secondarily to a great variety of bodily states; and, moreover, the disease occurs in several special forms which bear little etiologic relation to simple chronic bronchitis.

The vast majority of cases of simple chronic bronchitis are secondary to disease of the circulatory and bronchopulmonary systems. Imperfectly compensated heart disease, resulting in varying degrees of stasis in the lesser circulation, is very prone to initiate chronic bronchial irritation. Similarly, chronic bronchitis attends many chronic pulmonary diseases, notably phthisis, emphysema, and pneumonoconiosis.

Repeated attacks of acute bronchitis in elderly subjects are at times provocative of chronic bronchitis, these cases constituting the common winter coughs of these patients. Not infrequently emphysema is a concomitant affection.

Chronic bronchitis occurs in a hypertrophic and in an atrophic form. In the former there is proliferation of the mucous or goblet cells of the mucous membrane, which pour out a viscid grayish secretion containing innumerable desquamated epithelial cells mixed with bacteria. The lumen of the bronchus is diminished in numerous areas by hypertrophic thickening of the mucous membrane. In the atrophic form, on the contrary, the mucosa in certain areas of the bronchial distribution is thinned; the muscularis is replaced by fibrous connective tissue; and the weakened bronchial wall shows fusiform or saccular ectases.

In addition to the simple catarrhal form of the disease, chronic bronchitis occurs in several modified or special forms; notably as *purulent bronchitis,* as *fetid* or *putrid bronchitis,* and as *eosinophilic bronchitis.*

Purulent bronchitis, a form of extensive duration, is characterized by the expectoration of large quantities of purulent sputum of offensive odor, the foulness of the sputum, however, not approaching that of the putrid form of the disease. These cases sometimes exhibit a febrile movement which may simulate chronic ulcerative phthisis; and in other instances the temperature is septic, with colliquative sweats, causing excessive discomfort to the patient, and occasionally actually endangering life.

Fetid or *putrid bronchitis* is distinguished by the exceedingly foul odor of the expectoration, which contains characteristic small, gray pellets, composed of mucus, with bacteria, pus cells, fatty acid crystals and cellular detritus, Dittrich's plugs. The sputum is yellowish-gray, abundant, and upon standing separates into three layers: an upper of yellowish froth, a middle of transparent clear fluid, and a lower of purulent sediment. In addition to putrid bronchitis, purulent expectoration occurs in bronchiectasis, chronic ulcerative phthisis with cavitation, pulmonary abscess and gangrene, and empyema with pulmonary fistula. Putrid bronchitis is apt to produce aspiration pneumonia, pulmonary abscess, or gangrene of the lung, the patient exhibiting signs of sepsis, which are of grave prognostic import.

The ultimate result of persistent chronic bronchitis is pulmonary emphysema. The partial occlusion of the bronchioles combined with the paroxysms of violent cough are the prime factors in the production of this condition. However, the chronic bronchial irritation also results in peribronchial fibrosis, which by favoring interstitial sclerosis of the lung, also predisposes to bronchiectasis from traction. In other instances signs of right heart incompetence are manifested, as the result of interference with the pulmonary circulation, and in the absence of emphysema there is evidence of chronic venous stasis.

Physical Signs.—*Inspection.*—The subject of chronic bronchitis is liable to chronic shortness of breath, often attaining to the degree of actual dyspnea, upon moderate exertion. The paroxysms of violent cough in certain cases are suggestive of bronchial asthma. In the cases in which emphysema is coexistent the barrel chest of this disease with its limitation of expansion is noted, while in the circulatory group of cases there is apt to be cyanosis and edema from venous stasis.

Palpation.—Usually negative, palpation in cases associated with abundant secretion may reveal the presence of rhonchal fremitus of bilateral distribution. Vocal fremitus is little altered if indeed at all.

Percussion.—The percussion note in chronic bronchitis is often quite normal. However, in cases attended by emphysema the note is hyperresonant; whereas in cases of fetid bronchitis associated with bronchiectasis, dullness is encountered over large superficially filled ectases, the note changing to hyperresonance or tympany upon the expulsion of the contents of the cavities. Similarly there is in cases of cardiac origin not infrequently impairment of resonance over the bases posteriorly as the result of moderate pulmonary edema.

Auscultation.—Auscultatory signs in chronic bronchitis are abundant and very variable. In chronic bronchitis of extensive duration the respiratory murmur is harsh and by reason of the accompanying emphysema expiration is prolonged. Râles of all types are audible over both sides of the thorax, particularly over the bases posteriorly, at the inferior angle of the scapula, and anteriorly in the infraclavicular and mammary regions. In simple chronic catarrhal bronchitis large and small râles are in evidence.

Auscultation of the heart in chronic bronchitis yields variable and suggestive signs in many instances. In simple catarrhal bronchitis of comparatively brief duration the cardiac tones are unaltered; whereas in cases of cardiac origin and in cases which are associated with well-established emphysema, the second sound of the heart at the pulmonic area is accentuated; and in cases which are dependent upon chronic valvular disease the murmurs of the provocative lesions are audible. In cases of renal origin, on the contrary, the second sound at the aortic area is very frequently accentuated.

Diagnosis.—With a history of chronic cough recurring every winter, with râles distributed universally throughout both lungs, unattended by fever or loss of weight, in an elderly person who presents a thorax approaching the emphysematous type, the diagnosis of chronic catarrhal bronchitis is suggested. It should be borne in mind, however, that aortic aneurysm or mediastinal tumor are occasionally productive of chronic cough; but in these cases there is apt to be more or less pronounced stridor owing to unilateral vocal cord paralysis.

Putrid bronchitis with its foul sputum is apt to simulate the clinical picture of bronchiectasis. In most cases a differential diagnosis between putrid bronchitis and bronchiectasis with decomposition of the contents of the ectases may only be made when the latter disease forms cavities of sufficient size to give distinctive and definite physical signs or when revealed by radiography following instillation of iodized oil.

In dealing with a disease which is so frequently secondary to other pathologic changes in the thorax, the diagnosis cannot be considered complete until the causative lesion is in each case determined. In cases of cardiac origin auscultation of the precordia frequently reveals the presence of regurgitant lesions or signs of myocardial degeneration; while in cases dependent upon chronic renal disease the aortic second sound is accentuated and frequently there are signs of left ventricular hypertrophy. Again in cases of putrid bronchitis associated with pulmonary abscess or gangrene, careful physical examination will elicit confirmatory signs of the conditions.

It is of the utmost importance in cases of long standing chronic bronchitis to exclude the presence of chronic ulcerative phthisis, as the former is often part and parcel of the latter. In such event a careful search should be made for disease of the heart, arteries, or kidneys which might be responsible for the chronic bronchial inflammation. It should be recalled that a nontuberculous chronic bronchitis is seldom attended by febrile elevation, marked anemia, or loss of weight. Moreover, the physical signs in chronic bronchitis are bilateral, whereas in phthisis they are apt to be unilateral and to manifest a predilection for the pulmonary apices. In purulent and putrid bronchitis the sputum contains characteristic bodies in the form of Dittrich's plugs, Curschmann's spirals, and Charcot-Leyden crystals, whereas tissue shreds and elastic fibers, signs of tuberculous excavation of the lung, and the tubercle bacillus are absent from the sputum.

FIBRINOUS BRONCHITIS

Clinical Pathology.—Fibrinous coagula are formed in the smaller bronchial tubes under a variety of conditions. They have been noted in chronic disease of the lungs and heart; as a result of the inhalation of irritative gases, notably ammonia gas; as the result of infection of the bronchial mucous membrane by the pneumococcus or the Klebs-Loeffler bacillus, in the last instance the morbid process constituting a bronchial diphtheria. Following hemoptysis fibrinous masses are not infrequently expectorated, which may simulate those of fibrinous bronchitis.

Aside from these conditions, there are two conditions which may be classed as true fibrinous bronchitis with the expectoration of fibrinous moulds of the bronchial tubes amid paroxysms of dyspnea and cough; namely, the *acute fibrinous bronchitis* and the *chronic idiopathic fibrinous bronchitis.* The acute form of the disease is occasionally noted as a complication of the acute infectious fevers, notably in the course of scarlatina, measles, pneumonia, influenza, variola, tuberculosis, and typhoid fever. Fifteen cases of this type of the disease are reported by Bettman. Chronic idiopathic fibrinous bronchitis occurs as a chronic, recurrent malady, and the disease invariably attacks the same portion of the bronchial tree. In Bettman's collection of cases ten were accompanied by organic cardiac disease; fourteen developed during the course of pulmonary tuberculosis; five cases were associated with bronchial asthma; and four cases were attended by edema of the lungs.

The distinguishing feature of the disease is the expulsion from the bronchi and bronchioles of branching, fibrinous casts with the expectoration at the conclusion of a paroxysm of dyspnea and cough, which is relieved by the expulsion of the casts. The casts, which correspond in shape and size to the ramifications of the portion of the bronchial system which lodged them, vary in length and have been found to exceed ten centimeters in length. The body of the cast, which is composed largely of superimposed laminæ of fibrin with epithelial cells, leucocytes, bacteria, cellular detritus, and occasionally Charcot-Leydon crystals, corresponds in diameter with the lumen of the bronchial tube from which it was expelled.

Much confusion still remains as to the exact nature of the pathologic changes which occur in the mucous membrane of the bronchi in the affected areas. Weigert and Kretschy reasoned that the epithelial surface must desquamate in order to render possible the formation of the casts. Bettman, quoting Schittenhelm, describes a desquamative catarrhal inflammation of the alveoli with exudation into the alveoli, bronchioles, and smaller bronchioles.

Physical Signs.—*Inspection.*—During the paroxysms of dyspnea and cough which characterize the attacks the patient is usually orthopneic, the clinical picture simulating that of the paroxysm of bronchial asthma. The dyspnea is often relieved by the sudden copious expectoration, while in other instances the attack is attended by distressing cough with very scanty expectoration.

Palpation.—As a rule, palpation yields but limited data in this disease. However, when one of the larger bronchi is occluded by a cast, vocal fremitus is absent over the distribution of its radicles, and the expansion of the corresponding side of the thorax is diminished.

Percussion.—In the absence of concomitant disease of the lung, percussion of the thorax is usually negative in cases of fibrinous bronchitis. However, when a large bronchus is occluded the note is impaired over the area of the thorax corresponding to its distribution, to become resonant once more upon the expulsion of the offending cast. Moreover, it is occasionally possible to elicit a moderately hyperresonant note over the adjacent pulmonary tissues in the presence of bronchial occlusion, as a result of compensatory emphysema of the neighboring vesicles.

Auscultation.—Moist râles are frequently encountered, while occasionally the bruit de drapeau is audible. When present, it is of considerable diagnostic value. The respiratory murmur is absent over the distribution of an occluded bronchus, only to become reestablished with the expulsion of the cast and the restoration of the patency of the tube. The intensity of vocal resonance undergoes similar modifications under the same conditions.

Diagnosis.—Fibrinous bronchitis with its attacks of paroxysmal dyspnea may readily be confounded with bronchial asthma. The ultimate differential diagnosis depends upon the recognition of typical fibrinous casts in the sputum. While the physical signs are rarely typical of the disease, the presence of the bruit de drapeau is of great aid in diagnosis; and Andral has called attention to the importance of the transient abolition of the respiratory murmur over a circumscribed area of the thorax combined with normal pulmonary resonance. In all cases the presence of bronchial diphtheria and hemoptysis must be excluded.

BRONCHIECTASIS

Clinical Pathology.—Bronchiectasis is in the vast majority of cases a secondary disease, complicating previously existing disease of the bronchi, the lungs, or the pleura.

The dilatation may be produced by increased pressure exerted upon a weakened bronchial wall from within, or by traction exerted upon the bronchial wall from without. In chronic bronchitis of long standing, with its paroxysms of violent cough, combined with the accumulation of the abundant secretion in the purulent and putrid forms of the disease, the weakened bronchial wall is apt to yield and lead to bronchiectasis. The violent paroxysmal cough of pertussis may result similarly. Bronchial dilatation follows stenosis of the bronchi occurring as a result of syphilitic or tuberculous ulceration, and it has developed as a result of bronchial compression by mediastinal tumor or aneurysm.

The traction of adhesions upon the bronchial wall in chronic interstitial pneumonia, pulmonary syphilis, and fibroid phthisis is one of the most productive etiologic factors in the production of bronchiectasis. In these cases the cicatricial bands may pass from a thickened pleura to the affected bronchus, or they may pass from one bronchus to another. Bronchiectasis has followed the lodgment of foreign bodies in the bronchi. In these cases Cohn holds that the dilatation develops at the site of the foreign body as a result of irritation and consequent ulceration, and not distal to it as a result of stenosis. Bronchiectasis occasionally develops in connection with influenza, lobar or lobular pneumonia, chronic ulcerative phthisis, and pulmonary neoplasm. Moreover, bronchiectasis occurs in a congenital form, the "bronchiectasis universalis" of Grawitz.

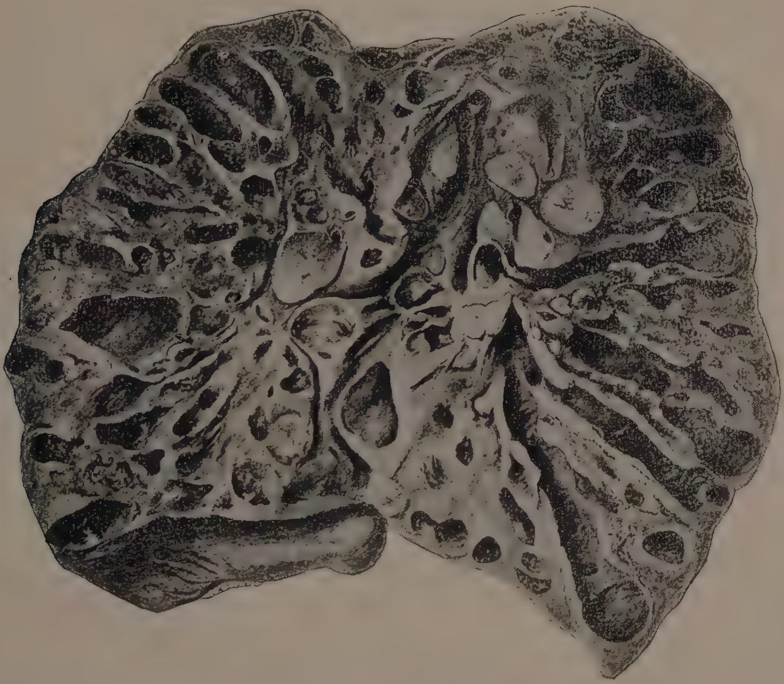

Fig. 276.—Sacculated bronchiectasis. (Pottenger, after Powell and Hartley.)

Autopsy experience has taught us that bronchiectasis is not an uncommon disease. The cases usually develop in young adults and in persons of middle age; and males are affected with the disease more frequently than are female subjects.

The bronchial dilatations are found most frequently in the right lung, in which they affect principally the bronchi of the middle and lower lobes. The dimensions of the ectases are variable, ranging from a moderate increase in the lumen of the tube to large cavities in which the bronchial communication is often obliterated. Two principal forms of dilatation are encountered; namely, the *saccular* and the *cylindrical*. Cylindrical dilatation is most frequently observed in connection with the smaller bronchial tubes; but it is also encountered in the larger bronchi; and the two forms, saccular and cylindrical, are not infrequently found in the same lung.

The pulmonary tissue adjacent to the larger ectases presents areas of compensatory emphysema, alternating with areas of sclerosis in which the pleura often participates. The number and distribution of the bronchiectatic areas are always variable; but as a rule a single large bronchiectasis occupies a position rather deep within the interior of the lung, whereas multiple small bronchiectases show a more superficial distribution.

The state of the mucous membrane of the dilatations varies. In certain instances scarcely altered, in practically all extensive dilatations it is thickened, with polypoid elevations upon the surface; while in cavities containing abundant secretion the mucosa is not infrequently ulcerated. The exudation from the walls of extensive bronchiectatic dilatations is usually purulent and abundant; but occasionally it is scanty and cheesy in consistence.

The expectoration, which represents the contents of the ectases, is gray or brown in color, mucopurulent in character, and in cases with large cavities is abundant and fetid. Upon standing, the sputum separates into three strata: an upper of brownish froth, a middle of semiopaque fluid, and a lower composed of granular sediment containing cellular detritus, bacteria, fatty acid crystals, leucocytes, and occasionally Charcot-Leyden crystals. The disease is occasionally, though rarely, attended by hemoptysis. Lime salts may deposit in bronchiectatic cavities. The dislodgement of these broncholiths may produce severe hemorrhage.

Bronchiectasis not infrequently results in emphysema, and these cases, constituting the ''dry cases'' of the disease, give the most favorable prognosis. In cases with large ectases and cavitation, associated with the collection of abundant secretion, abscess or gangrene of the lung is apt to supervene. Abscess of the brain has been noted in connection with bronchiectasis, usually upon the side corresponding to the site of the dilatation.

Physical Signs.—*Inspection.*—The presence of small bronchiectases produces no characteristic physical signs. In the fully established case of bronchiectasis, however, in which the dilatations have attained considerable size, there is impairment of expansion of the corresponding side of the thorax and occasionally retraction of the affected side with drooping of the shoulder. The decubitus of the patient, while variable, in advanced cases is suggestive; as the patient usually prefers to lie upon the affected side, and a change of posture is frequently followed by a severe paroxysm of cough due to shifting of the contents of the cavities.

Clubbing of the finger tips from enlargement of the terminal phalanges is present in many cases of long-standing bronchiectasis.

Palpation.—In the presence of a large bronchiectatic cavity with patent bronchial communication, situated superficially in the thorax, vocal fremitus is very markedly increased when the cavity is empty, to become abolished over the same area when the cavity is filled with fluid.

Percussion.—The findings upon percussion in bronchiectasis are variable, depending upon whether the cavity under investigation is empty or is filled with secretion. If the cavity contains fluid, even though it has a patent bronchial outlet, the percussion note is dull; whereas, if the cavity is empty, it yields tympany or a cracked-pot sound upon percussion. In suitably situated cavities all of the signs of pulmonary cavity, such as Wintrich's change of sound, Friedreich's respiratory change of sound, and the change of sound of Gerhardt, may be elicited.

In every case in which the signs of cavitation are evanescent, present and absent at successive examinations, the possibility of bronchiectasis should be borne in mind, as these cavities fill with secretion which masks all physical signs, and then the signs of cavitation reappear with the evacuation of the contents of the dilatations.

Auscultation.—In cases of bronchiectasis which are of relatively short duration, auscultation yields only the signs of chronic bronchitis, puerile breath sounds, and râles. If, however, a rather large dilatation be properly situated with reference to its bronchial communication, amphoric breathing is encountered. If a large dilatation is situated near the periphery of the lung, and if it has a free bronchial outlet, vocal resonance is very greatly exaggerated, perhaps to the extent of affording bronchophony or whispering pectoriloquy.

The x-ray has made it possible to diagnose many more cases of bronchiectasis than was possible by physical signs alone. Iodized oil (lipiodol) is instilled into the trachea and allowed to slowly travel into the smaller bronchi. The x-ray shadow following this instillation reveals the dilated bronchi and bronchioles very clearly.

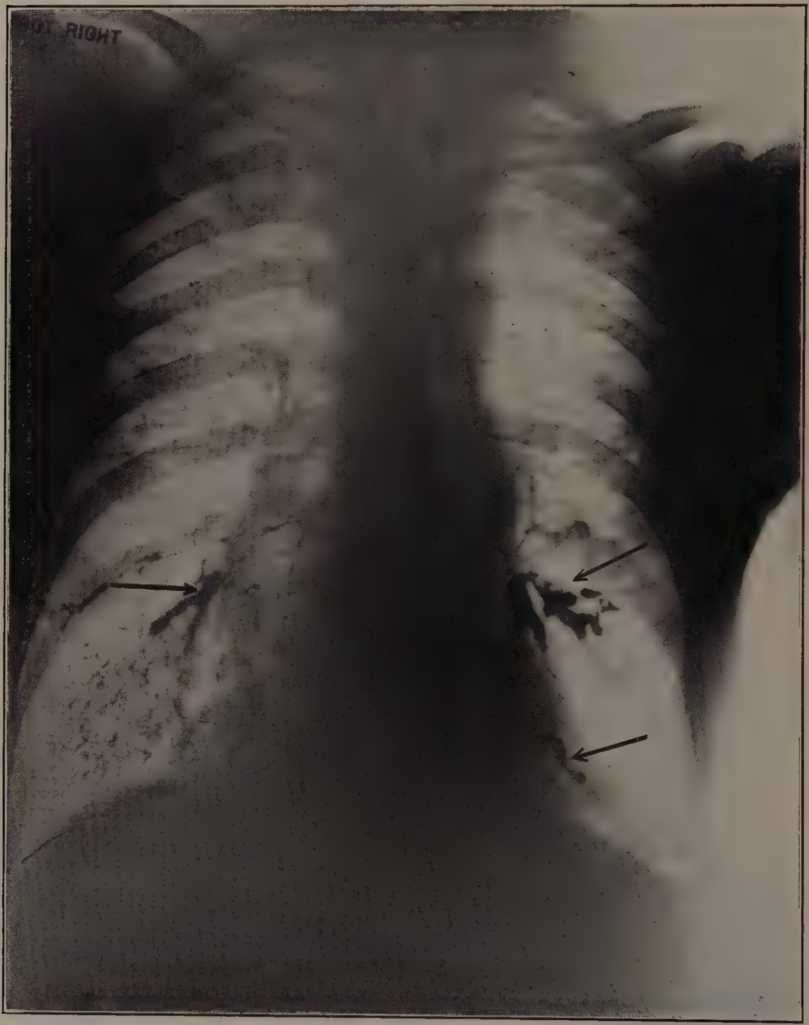

Fig. 277.—Radiograph of a case of bronchiectasis, showing sacculations filled with iodized oil (indicated by arrows).

Diagnosis.—Cases of moderate bronchiectasis are distinguished from chronic bronchitis with difficulty, of which disease indeed it is often a sequel. In well-established cases, however, in which more extensive organic change has occurred in the bronchial system, the expectoration of a copious amount of mucopurulent sputum at one time, followed by an absence of expectoration for several hours, is suggestive of bronchiectasis; and when in addition signs of cavitation can be elicited over the base of the lung, the presence of bronchiectasis may be assumed.

Clubbing of the fingers and changes in the lower extremities approaching those of pulmonary osteoarthropathy are particularly suggestive of bronchiectasis.

Differential Diagnosis.—The most important point in differential diagnosis is the possibility of mistaking bronchiectasis for chronic ulcerative phthisis with cavity formation. However, cavities in the latter disease are usually situated in the pulmonary apex, a region of the lung which is rarely invaded by bronchiectasis. Moreover, percussion of the lung adjacent to the cavity in bronchiectasis is apt to yield hyperresonance due to compensatory emphysema, whereas in phthisis similar percussion elicits dullness of consolidation, which at this advanced stage of the disease is usually demonstrable also upon percussion of the opposite lung. In phthisis the sputum is raised at frequent intervals and is apt to contain the tubercle bacillus and elastic fibers. Moreover, chronic ulcerative phthisis produces characteristic deformity of the thorax with fever, anemia, and night sweats, while the course of the disease is progressively downward to a fatal termination. In bronchiectasis, on the contrary, unless it develops upon a tuberculous basis, the physical signs persist for a long period, while the patient remains in a comparatively good state of health.

Empyema with rupture into a bronchus is attended by the sudden expectoration of a large amount of purulent sputum; but instead of signs of cavity over the lower lung, there is the dullness of the original empyema, with the probable presence of Grocco's sign.

Pulmonary abscess and gangrene, which may simulate bronchiectasis, are excluded by their more rapid evolution, and by the presence of signs of sepsis. Putrid bronchitis is excluded by its bilateral distribution.

The x-ray following the instillation of iodized oil usually clinches the diagnosis.

BRONCHIAL ASTHMA

Clinical Pathology.—Bronchial or spasmodic asthma consists essentially of a paroxysmal dyspnea which is almost entirely expiratory in type, the subject of the disease during the paroxysm being unable adequately to expel the tidal air from the lungs. Bronchial asthma has nothing in common with the so-called cardiac asthma or renal asthma.

Numerous theories have been advanced in the effort to explain the cause of the asthmatic paroxysm. It has been suggested that the attack is initiated by a sudden spasm of the circularly disposed bronchial musculature; and that the obstacle to the egress of the tidal air from the lungs is due to narrowing of the lumen of the bronchioles by temporary turgescence of the mucous membrane. Curschmann held that the underlying cause is to be found in inflammation of the smaller bronchioles, the so-called *bronchiolitis exudativa* of this author. Spasm of the diaphragm has likewise been advanced as a probable cause of the paroxysm.

Recent clinical and experimental work has established the fact that in true allergic asthma the patient is hypersensitive to some form of protein, and that the asthmatic paroxysm is to be attributed to inhalation or ingestion of the offending protein. These have been found to include various cereal grains, the protein of egg and meats, as well as the pollens of various plants and the emanations from numerous animals. In yet other cases the patient is hypersensitive to the protein of bacteria which are harbored in focal infections.

In the cases which have been subjected to autopsy the ciliated epithelium has been found in a state of desquamation, with bronchial congestion and exudation rich in eosinophilic cells.

The circulating blood during the paroxysm of bronchial asthma contains an excess of eosinophiles, these cells representing 25 per cent to 35 per cent of all the leucocytes.

The sputum in bronchial asthma is characteristic of the disease. In the early stages it is scanty and very tenacious, containing Curschmann's spirals and Charcot-Leyden crystals. Macroscopically Curschmann's spirals are white or yellow, assuming the form of twisted threads or of small balls. The length of the spiral rarely exceeds half an inch, but it may exceed two inches in certain instances. Under the microscope they appear as mucous threads containing a clear, central fiber around which are wound numerous fine fibrils. Charcot-Leyden crystals occur as colorless, pointed, octohedral crystals, the average length of which is about three times the diameter of a red blood cell. They are frequently absent from the freshly expectorated sputum, but they appear after it has stood for a short time.

During the later stages of the attack these two pathognomonic elements of the sputum disappear, the expectoration becoming more abundant and mucopurulent in character.

Chronic bronchitis is frequently a concomitant affection in subjects of bronchial asthma; and the repeated paroxysms of the disease, with their typical expiratory dyspnea, tend to the production of emphysema and bronchiectasis.

Physical Signs.—*Inspection.*—During the paroxysm of bronchial asthma, which is very abrupt in onset, dyspnea of the expiratory type dominates the other physical signs. There is little if any increase in the number of respirations; and indeed, owing to the great prolongation of expiration, the number may be actually diminished. Inspiration is short and powerful, but adds little to the degree of expansion of the thorax, which remains fixed in a position of relatively full inspiration. Bamberger describes the inspiratory phase as that of normal inspiration with the participation of the accessory muscles of respiration in the act. Expiration, which lasts three to four times as long as inspiration, follows the latter promptly without the intervention of the normal pause between these phases, the accessory muscles of expiration being called into play in the attempt to empty the thorax.

Litten's sign may be sharply defined but shortened during the early part of the attack and entirely absent as the lungs become more engorged and the diaphragm flattened.

During the course of the paroxysm the patient becomes orthopneic; the cervical veins become engorged; and the lips, face, and hands are apt to become cyanotic. The thorax is large and semifixed in the position of inspiration; the diaphragm is depressed; and the moderate degree of expansion during inspiration is exerted largely in the vertical direction, the thorax rising and falling *en masse* much in the same manner as in hypertrophic emphysema.

Palpation.—During the paroxysm vocal fremitus, if its intensity can be determined, is diminished as a result of the rarefaction of the transient acute vesicular emphysema or of bronchial obstruction by turgescence of the bronchial mucosa or of spasm of the bronchial muscles. Rhonchal fremitus is present to a striking degree, and is bilateral in its distribution. It may be felt over one given area, later disappearing at this point and reappearing at another portion of the chest. The cardiac impulse is often masked by the concomitant emphysema of the anterior pulmonary borders. Pulsation is frequent in the cervical vessels and in the episternal notch.

Percussion.—The quality of the percussion note in bronchial asthma varies from a moderate hyperresonance during mild attacks to a note which closely approximates tympany in severe attacks in emphysematous subjects. The limits of pulmonary resonance are extended in all directions, upward in the supraclavicular regions, downward encroaching upon the hepatic and splenic dullness, and anteriorly, causing diminution in the area of cardiac dullness. In advanced cases, which are associated with marked emphysema, the area of cardiac dullness is frequently displaced downward. The respiratory excursion of the pulmonary borders is strikingly diminished in these subjects.

Auscultation.—The vesicular murmur is obscured by numerous loud, sibilant and sonorous râles, which are so pronounced as frequently to be audible at some distance from the patient. Late in the paroxysm, after the bronchial secretion has become freely established, the dry râles give place to numerous moist and bubbling râles. Inspiration is frequently entirely inaudible, whereas the expiratory phase of the vesicular murmur is greatly prolonged and is punctuated with numerous râles.

Alexander in his recent monograph finds little to be added to the physical picture.

Diagnosis.—Owing to the paroxysmal character of the asthmatic attack, with its well-marked expiratory dyspnea dotted with râles, the overdistention and relative fixation of the thorax, and the characteristic elements of the sputum, a diagnosis of bronchial asthma is often readily made. However, it is not infrequently necessary to differentiate the disease from attacks of dyspnea arising from other causes. Prominent among the latter is dyspnea due to laryngeal obstruction from spasm or edema of the glottis. In this case the dyspnea is distinctly of the inspiratory type, often with stridor, and as a rule is attended by marked respiratory movement of the larynx. There is often aphonia; the thorax is of normal size; and there is apt to be inspiratory recession of the lower intercostal spaces. The absence of râles in the presence of laryngeal obstruction is in marked contrast to the numerous râles of the asthmatic attack.

The x-ray shows increase in size of bronchial shadows with decrease in density of lung parenchyma.

Differential Diagnosis.—Bronchial compression by enlarged glands, tumors, or aneurysm, and stenosis resulting from the ingestion of foreign bodies are to be excluded before a diagnosis of bronchial asthma is to be made upon the presence of paroxysms of severe dyspnea.

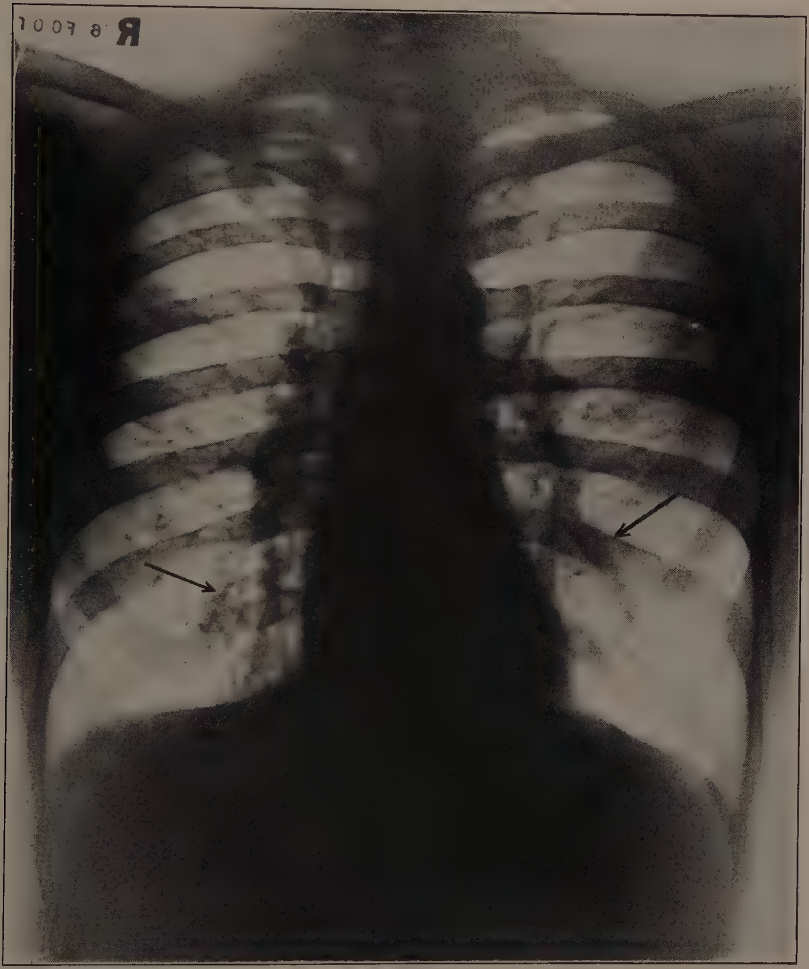

Fig. 278.—Radiograph of a case of bronchial asthma with increased density of the bronchial shadows (indicated by arrows).

Hypertrophic emphysema, chronic bronchitis, and bronchial asthma are often differentiated with difficulty, and indeed they are frequently concomitant diseases. However, in pure hypertrophic emphysema the expiratory prolongation and the attendant physical signs are constant and are not paroxysmal in their manifestation. Moreover, in emphysema the vital capacity of the lungs is permanently diminished, whereas in bronchial asthma in the intervals between the paroxysms this capacity closely approximates the normal, to become abruptly impaired during the asthmatic attack. Moreover, the microscopic examination of the sputum in bronchial asthma reveals the presence of characteristic elements. As to chronic bronchitis and bronchial asthma, the former may be assumed to be present when in the intervals between the paroxysms physical signs of moist bronchial inflammation are persistently discovered upon successive examinations. Cardiac asthma, while a paroxysmal dyspnea, shows none of the characteristics of bronchial asthma.

The response of sufferers from bronchial asthma to the administration of adrenalin is of great diagnostic value.

BRONCHIAL STENOSIS

Clinical Pathology.—Diminution of the lumen of a bronchus arises from intrabronchial and from extrabronchial causes. Intrabronchial stenosis occurs with tracheal and bronchial diphtheria, as the result of the development and growth of polypoid tumors of the mucous membrane, as a consequence of turgescence of the mucosa following the inhalation of irritating gases, in cicatricial formations following syphilitic ulceration, and in the presence of foreign bodies. Below the bifurcation the bronchial tree is liable to compression from aortic aneurysm, pericardial effusion, or an excessively hypertrophied heart, pulmonary tumor, or enlarged bronchial glands. Aneurysm of the ascending aorta usually compresses the right bronchus, whereas aneurysm of the arch or of the descending aorta causes compression of the left bronchus.

The ultimate effect of the stenosis upon the lungs varies with the degree and with the site of the obstruction. Stenosis above the tracheal bifurcation results in deficient, but prolonged expansion of the lungs. Total and permanent obstruction of a principal bronchus results in ultimate collapse of the lung which it supplies. Complete stenosis of a bronchial tube of the second or third order results in atelectasis of a considerable area of a lung; while stenosis of several of the small bronchi exerts little influence upon the lung as a whole, as the area of atelectasis is compensated by vicarious expansion of the adjacent pulmonary lobules.

Upon complete stenosis of any portion of the bronchial system, the air in the infundibula supplied by the affected tube is gradually absorbed and the pulmonary lobules collapse, constituting the so-called *obturation atelectasis.* The lobules immediately adjacent to an area of atelectasis are compensatorily emphysematous, and by their vicarious expansion and increased aerial content mask the physical signs which would naturally be produced by the area of atelectasis.

Physical Signs.—The physical signs of bronchial stenosis vary with the site and the degree of the obstruction. In stenosis of a principal bronchus the physical manifestations of the condition are unilateral, but may affect an entire lung. In stenosis of a small bronchus physical signs are slight in degree or are entirely absent.

Inspection.—In stenosis of a principal bronchus expansion is limited over the side of the affected lung. Bronchoscopy is often of service in the detection of stenosis, revealing the site and the degree of the obstruction.

Palpation.—Vocal fremitus is diminished or absent over the distribution of the obstructed tube, the alteration in intensity being unilateral in bronchial obstruction.

Percussion.—In stenosis of a principal bronchus the percussion note is dull over the area of distribution of the bronchus, or over the entire lung. In minor degrees of stenosis the note is scarcely changed, and indeed it may be hyperresonant as a result of the compensatory emphysema of the infundibula adjacent to the area of atelectasis.

Auscultation.—Upon auscultation over the area of an occluded bronchus of considerable magnitude the respiratory murmur is feeble or is entirely abolished, while over the opposite lung it is exaggerated or puerile. Coarse, sonorous râles are occasionally to be elicited over the site of the obstruction. Vocal resonance is abolished over the affected lung in the presence of obstruction of a principal bronchus; but it is little affected in minor degrees of stenosis.

Diagnosis.—The presence of inspiratory dyspnea, sibilant and sonorous râles, confined to a circumscribed area, unilateral deficiency of expansion and dullness over an entire lung, are signs suggestive of obstruction of a principal bronchus.

X-ray may reveal the cause of obstruction and may accurately map out the portion of the lung supplied by the obstructed bronchus.

CHAPTER XXXVII

CIRCULATORY DISTURBANCES OF THE LUNGS

PULMONARY CONGESTION

Clinical Pathology.—Congestion of the lungs occurs in two forms; namely, as *active congestion,* and as *passive congestion.*

Active congestion of the lungs occurs in the early stages of inflammation of these organs, as in the period of engorgement of lobar pneumonia. But active congestion of the lungs frequently occurs in conditions which do not attain the gravity of the first stage of lobar pneumonia; notably after the inhalation of irritating gases, of hot or cold air; and it occurs as a collateral congestion due to disease of an adjacent area of the lung. Active pulmonary congestion while not usually dangerous in itself, has in rare instances terminated fatally. Postmortem the lung in active congestion is enlarged, is deep red, its consistence is increased, yet the lung is crepitant and will float when placed in water.

Passive congestion of the lungs occurs in two forms; namely, as *mechanical congestion,* and as *hypostatic congestion* of the lungs.

Mechanical congestion of the lungs results from an obstacle which is interposed to the free return of blood from the lungs to the heart. The most common cause operating in this manner is mitral regurgitation, while a less frequent cause is a tumor pressing upon the pulmonary veins. Mitral stenosis and aortic insufficiency and stenosis operate similarly to produce mechanical congestion of the lungs.

Hypostatic congestion of the lungs is encountered in adynamic and asthenic states, particularly in elderly subjects who have been long in the recumbent posture during a continued fever or chronic wasting disease. The congestion in this instance is localized to the posterior and inferior portions of the lungs, and is largely dependent upon general asthenia and relaxation of the pulmonary vessels. That it is not entirely due to the supine posture is evinced by the fact that it occurs only in subjects who are weakened by disease.

In passive congestion of the lungs the vessels of the lung are dilated and the interalveolar septa are distended with fluid, while the alveolar spaces contain serous fluid, desquamated alveolar cells containing blood pigment, and a few leucocytes and red blood cells.

Physical Signs.—*Inspection.*—In pulmonary congestion the respiratory movements of the thorax are hurried but are limited in amplitude, the degree of thoracic expansion being below the normal degree. In acute congestion the face exhibits a variable degree of cyanosis; the expression is that of great anxiety; and the alæ nasi move with respiration.

Palpation.—In active congestion palpation reveals a moderate exaggeration of vocal fremitus, which is most readily detected over the bases posteriorly. In passive congestion, on the contrary, there is diminution of the fremitus over this area of the thorax.

Percussion.—In active congestion of the lungs the percussion note is apt to be slightly hyperresonant, as a result of the increased tension of the pulmonary tissues; whereas in passive congestion there is impairment of resonance over the bases posteriorly, owing to the gravitation of the blood to these regions of the pulmonary system.

Auscultation reveals in active congestion bronchovesicular breathing; and, in cases of passive congestion shows in addition the valvular lesion which is responsible for the congestion. In each instance the pulmonic second sound is accentuated as a result of the increased tension in the pulmonary circulation. In passive congestion moist or bubbling râles are not infrequently audible over the bases posteriorly.

Diagnosis.—Dyspnea with anxious facies, in conjunction with a history of violent physical exertion, or exposure to irritant vapors or in the presence of active pulmonary disease, with limited excursion of the thorax is suggestive of acute pulmonary congestion, particularly if the acute symptoms develop abruptly without warning. The presence of dullness, impaired vocal fremitus, and moist or bubbling râles over the bases posteriorly in an asthenic or bed-ridden patient, is suggestive of passive congestion of the lungs; while, if a valvular lesion of the heart is coupled with the respiratory findings, the diagnosis is yet more probable.

PULMONARY EDEMA

Clinical Pathology.—Edema of the lungs occurs in an acute and a chronic form and as general and local edema.

Acute pulmonary edema complicates the acute infectious diseases, as during the course of lobar or lobular pneumonia, scarlatina, influenza, acute rheumatic fever, variola, and typhoid fever. Less frequently it has developed during pregnancy and in hysterical patients.

Chronic pulmonary edema occurs as a result of cardiac weakness during the course of myocarditis, valvular disease, chronic interstitial nephritis, or general arteriosclerosis.

General pulmonary edema involves the whole of both lungs, the manifestations of the stasis being more marked in the bases as a result of gravitation of the fluid toward the dependent portions of the lungs. The lung of general pulmonary edema is sodden, and heavy; the alveoli of the bases contain serous fluid, while the interalveolar walls are thickened and edematous. The lung pits upon pressure, and upon section serous fluid exudes from the surface of the section. The alveoli contain numerous desquamated alveolar epithelial cells which contain blood pigment, the so-called "heart-failure cells."

Local pulmonary edema occurs as a collateral edema about foci of active pulmonary inflammation in the course of pneumonia, phthisis, and pulmonary infarction. In these cases the signs of the primary affection in the main obscure those of the local edema.

General pulmonary edema is attended by the expectoration of abundant, clear, thin, frothy sputum, which is raised in large quantities.

Physical Signs.—*Inspection.*—The subject of acute pulmonary edema is suddenly seized with dyspnea, which rapidly progresses to orthopnea, with anxious facies, and frequently with cyanosis of the lips and buccal mucosa. Thoracic expansion is diminished in extent, but is increased in frequency. There is constant, harassing cough, the cough of the wet lung, which is attended by the raising of abundant, frothy expectoration.

Palpation.—Vocal fremitus is diminished in intensity over the bases of the lungs, while rhonchal fremitus, the tactile equivalent of the numerous moist râles which are present, is well brought to the fore. The skin of the extremities is moist and cold. The pulse is rapid, with diminution in the volume and force of the waves.

Percussion.—The resonance of the percussion note is impaired over the bases posteriorly, whereas over the anterior surface of the chest in the infraclavicular and mammary regions skodaic resonance is not infrequently encountered.

Auscultation.—The respiratory murmur over the infraclavicular and mammary regions is commonly bronchovesicular, while over the bases vesicular respiration is masked by numerous moist and bubbling râles. The heart sounds are increased in frequency, and exhibit a diminution in their intensity, save that the pulmonic second sound is frequently moderately accentuated. In extreme cases of general pulmonary edema, associated with cardiac dilatation, embryocardia is frequently present late in the course of the disease.

Diagnosis.—The diagnosis of pulmonary edema rests upon the occurrence of dyspnea, frequently amounting to orthopnea, accompanied by impairment of resonance and moist râles over the pulmonary bases, in a subject with an obstacle to the return of blood to the left heart or in an asthenic state from an acute infection or the subject of chronic nephritis or arteriosclerosis.

Pulmonary edema is differentiated from bronchial asthma by the fact that in the former the dyspnea involves both phases of the respiratory cycle, and by the essential characteristics of the expectoration in the two affections.

PULMONARY INFARCTION

Clinical Pathology.—Infarction of the lung occurs as a result of occlusion of one or more terminal branches of the pulmonary arteries. The condition develops most frequently in connection with valvular heart disease, particularly with acute or chronic endocarditis. Pulmonary infarction is almost invariably of the hemorrhagic type.

The areas of infarction are situated most frequently in the lower lobes, and involve the right lung more frequently than the left. Usually of limited extent, a pulmonary infarct may become very extensive, involving the greater part of a lobe of the lung. The infarcts are commonly situated at the periphery of the lung; they are wedge-shaped; and the base of the wedge is directed toward the free surface of the lung. When recent, the areas of infarction are dark red upon section, resembling a blood clot. Later, as organization progresses and the hemoglobin is partially removed by phagocytes, they assume a denser consistence and a yellowish color. Eventually, in the absence of pyogenic organisms, the infarct undergoes organization, leaving a puckered scar at the site of infarction. The pleura overlying an area of infarction exhibits signs of local inflammation.

Microscopically the alveoli of the lung in the area of infarction are filled with erythrocytes in various stages of disintegration. The vessels of the alveolar walls are likewise filled with red blood cells and in places are apt to exhibit thrombus formation.

The ultimate termination of a pulmonary infarct depends upon the character of the causative embolus. If the embolus which occluded the terminal artery is noninfectious, the infarct gradually undergoes organization, and eventually is converted into a mass of cicatricial tissue at the site of infarction. If, on the contrary, the embolus is of septic origin, the area of infarction is frequently the point of incidence of a pulmonary abscess or of pulmonary gangrene. It is probable that in the case of large areas of infarction, involving the major portion of a lobe of the lung, partial resolution analogous to that which follows the consolidation of lobar pneumonia ensues.

Infarction of the lung is not infrequently attended by the expectoration of viscid, mucoid sputum containing numerous erythrocytes, and occasionally by frank hemoptysis.

Physical Signs.—The signs which are referable to pulmonary infarction are variable, depending as they do upon the number, the size, and the distribution of the infarcts. In the case of small infarcts and of infarction of central portions of the lung, physical signs may be entirely lacking; or the only signs elicited may be referable to the associated pleural inflammation, and may be in no way distinctive of infarction.

Inspection.—In cases of extensive pulmonary infarction the respiratory excursion of the thorax is limited upon the side of the disease. In addition, the subject is apt to exhibit moderate or severe dyspnea, with anxious facies and occasionally hemoptysis.

Palpation.—Palpation may show increased vocal fremitus if the area of infarction is of considerable extent and is situated near the periphery of the lung; whereas, if the infarct is centrally placed, near the root of the lung, no alteration of vocal fremitus is demonstrable. When a peripheral infarct overlies a principal bronchus, vocal fremitus is markedly exaggerated.

Percussion.—Over large infarcts dullness is elicited upon percussion; whereas in the case of a large infarct which is superimposed upon a principal bronchus, the tympany of the bronchus is engrafted upon the dullness of the percussion note. Gerhardt calls attention to the frequency of pulmonary infarction upon the right side in the area of the lung lying between the vertebral column, the diaphragm, and the angle of the scapula, whence the importance in this disease of signs of consolidation over this area.

Auscultation.—In suitably situated infarcts with reference to a main bronchus, loud, bronchial or tubular breath sounds are elicited upon auscultation, as well as râles which are transmitted from the bronchial tube. In cases of multiple infarcts, or of deeply seated infarction, the breath sounds are at the most bronchovesicular, and in many instances they are frankly vesicular.

Pleural friction is frequently to be elicited over areas of superficial infarction; and indeed in the case of infarction of limited extent, involving the peripheral portion of the lung, this is occasionally the only sign of the disease.

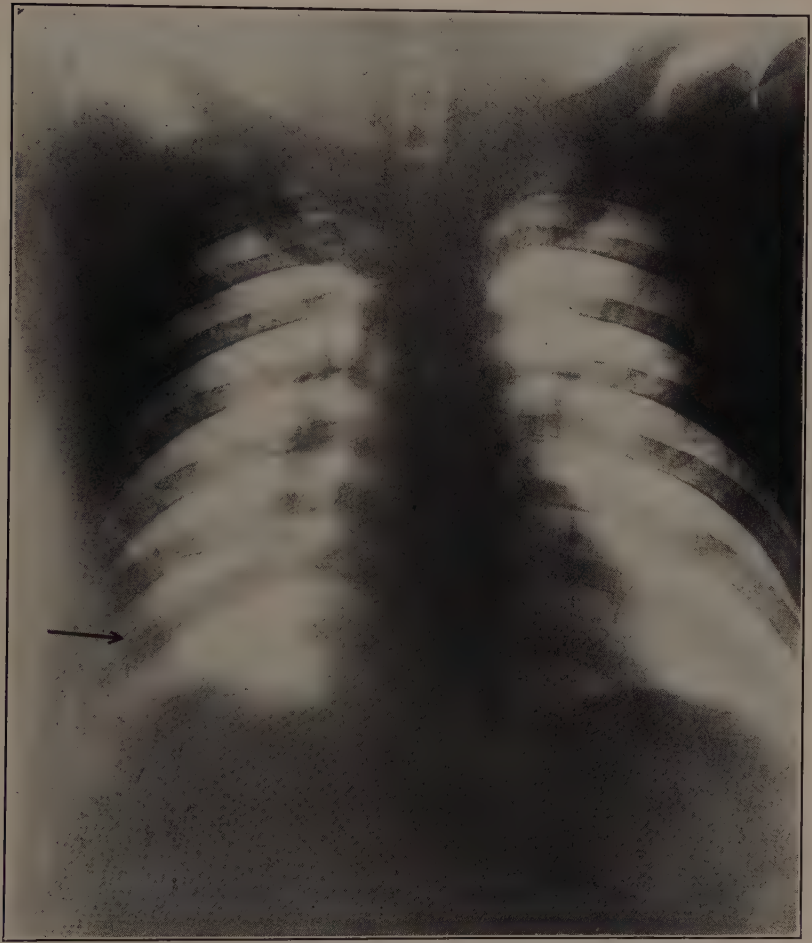

Fig. 279.—Radiograph of pulmonary infarction. Note wedge-shaped area of density at base of the right lung (indicated by arrow).

X-ray examination sometimes reveals a typical wedge-shaped area of increased density. If the area is deeply situated the x-ray may yield the only positive sign.

Diagnosis.—The physical signs of infarction of the lung are essentially those of pulmonary consolidation, and the diagnosis of infarction is made with difficulty. When signs of consolidation are elicited in a patient suffering with valvular heart disease, from which pulmonary embolism might arise, infarction of the lung becomes a possibility; and, when the physical signs of consolidation are attended by hemoptysis, pulmonary infarction may be assumed to be present. As the area of infarction is frequently situated in the lower lobe of the right lung posteriorly, the physical signs of consolidation simulate closely those of lobar pneumonia.

An infarct of moderate size occupying a central portion of the lung produces no physical signs by which a diagnosis of infarction may be made. A limited, superficial infarct, on the contrary, is apt to produce a local friction rub, which is apt to be mistaken for a simple pleurisy. Septic infarction, if extensive, is attended by signs of septic intoxication, with the supervention of signs of pulmonary abscess or of pulmonary gangrene.

CHAPTER XXXVIII

DISEASES OF THE LUNGS

LOBAR PNEUMONIA (CROUPOUS, OR FIBRINOUS PNEUMONIA)

Clinical Pathology.—Lobar, croupous, or fibrinous pneumonia is a specific inflammation of the lung, associated with a variable degree of constitutional reaction and toxemia. The designation "lobar" indicates that the usual tendency of the disease is to involve a lobe or a more extensive area of the lung, while the terms "croupous" and "fibrinous" have reference to the character of the inflammatory exudate.

The specific cause of lobar pneumonia is the pneumococcus, or Diplococcus pneumoniae, first discovered in the sputum of pneumonia patients by Sternberg and Pasteur in 1880, and recognized as the causative organism of the disease by Fraenkel in 1884. The pneumococcus may be found in pure culture in the sputum of pneumonia patients, or, as is more frequently the case, associated with the streptococcus, staphylococcus, Friedländer's bacillus, or other bacteria.

Recent studies in immunity and agglutination have revealed the fact that all pneumococci may be grouped in four classes or types. Of these several types of the organism, Type I is the cause of approximately 33 per cent of the cases of lobar pneumonia, Type II of a similar number, Type III of 10 per cent to 15 per cent, while the organisms of Type IV are responsible for about 20 per cent of the cases of the disease. Pneumococci of Types I, II, and III are not normally present in the buccal and nasal secretions; but they are frequently demonstrable in the throats of pneumonia patients and of carriers during epidemics, whereas those of Type IV, a group which is represented by a heterogeneous collection of pneumococci, are frequently demonstrable in the nasopharyngeal secretions of persons in health.

The mortality from infection with Type I and Type II of the pneumococcus is about equal, approximating 25 per cent to 30 per cent, whereas that from Type III exceeds the mortality from other types, usually reaching 50 per cent. The mortality from

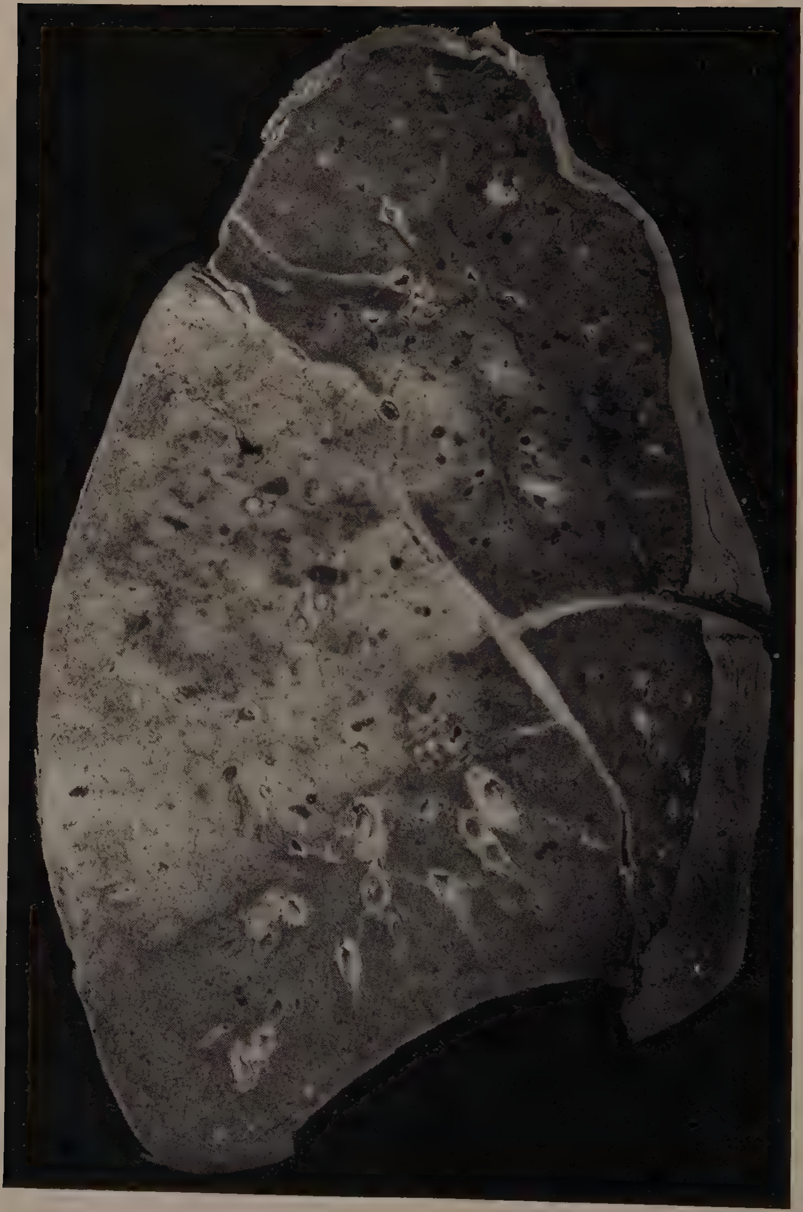

Fig. 280.—Lobar pneumonia, showing extension of gray hepatization from lower to upper lobe through a defect in the septum separating the two lobes. (From Opie, Blake, Small, Rivers.)

the group of organisms represented by Type IV averages 12 per cent of the subjects attacked by these strains.

Lobar pneumonia is prevalent at the extremes of life, affecting especially children and elderly persons; but no age is exempt from the disease. Among adults, males are more frequently attacked by the disease than are female subjects; but during childhood the disease shows no definite predilection for either sex.

The disease exhibits a distinct seasonal incidence, the majority of the cases occurring during the late winter and early spring months, although isolated cases are noted at all seasons. The onset of acute symptoms frequently follows exposure to inclement weather, suggesting that reduction of the patient's resistance to infection probably permits pneumococci harbored in the respiratory passages to initiate the acute inflammation of the lung (autoinfection). Postoperative lobar pneumonia is probably due to similar causes resulting from chilling of the body incident to anesthesia and operative procedures. Autoinfection seems to be more frequent in the case of Type IV than with other types of pneumococci.

There is sufficient evidence available, however, to justify the statement that lobar pneumonia can be directly communicated from a subject of the disease, when overcrowding or unsanitary conditions prevail. Numerous epidemics of the disease are on record in which this was the only apparent method of spread of the disease. The infecting organism in these cases is usually of Types I, II and III.

The morbid pulmonary changes incident to lobar pneumonia present four fairly well-defined stages; namely, the stage of engorgement, the stage of red hepatization, the stage of gray hepatization, and, if the patient recovers from the disease, the stage of resolution. However, these stages of the inflammation are not invariably recognizable as distinct and separate entities; and it is not uncommon to find one stage more or less blended with another.

The stage of engorgement is of brief duration, rarely exceeding twenty-four hours, as it is early followed by hepatization of the lung. During the period of active hyperemia which constitutes the stage of engorgement, the lung is dark red in color, and firm to the touch; but it is still crepitant, and the lung will float if placed in water. Microscopically during this stage the capillaries are distended with erythrocytes; the alveolar walls are thickened; and the alveolar spaces contain a variable number of erythrocytes, leucocytes, and desquamated epithelial cells.

During the stage of red hepatization the pulmonary tissue in the portion of the lung which is the site of the disease is solid, firm, and devoid of air. The lung is slightly enlarged, and frequently the surface presents indentations corresponding to the ribs with which it is in contact. Upon section, the surface of section is dry, reddish or brown in color, and very friable. Upon scraping the cut surface with the knife, small fibrinous plugs are apt to come away from the cut surface of the lung. The lung is not crepitant in the area of the disease; and a section of the lung will sink when placed in water. Microscopically the alveoli are filled with a dense, fibrinous exudate, which contain erythrocytes, leucocytes, bacteria, and desquamated epithelial cells, embedded and entangled in a matrix of fibrin, the erythrocytes predominating during this stage of the disease.

In the stage of gray hepatization the pulmonary tissue loses its reddish color upon section, the surface of section presenting a gray or grayish-white appearance. Section shows a moister surface than in the preceding stage, and but few fibrinous plugs come away upon scraping the surface with the knife. Microscopically, polymorphonuclear leucocytes predominate the picture, though a variable number of erythrocytes, as well as desquamated epithelial cells and bacteria are present in the fibrinous mass. However, notwithstanding the partial clearing of the alveoli, the lung is still noncrepitant, and a section of the diseased area will sink when placed in water.

During the stage of resolution the fluid is drained from the lung by the lymphatics; the débris is removed by phagocytes, and is also expectorated with the sputum; and the lung gradually resumes its normal structure. The area of disease once more becomes crepitant, and a section floats when placed in water.

The expectoration during the active stage of lobar pneumonia is thick and viscid, and of a brownish color, the so-called "prune juice sputum." Frequently the viscosity is so great that the sputum will not separate from its container when the latter is inverted.

During the inflammation of the lung in lobar pneumonia the pleura is practically always involved over the area of consolidation, becoming coated with a variable quantity of fibrinous exudate, and not infrequently pouring out a moderate amount of serous fluid into the pleural cavity. However, in central pneumonia, in which the consolidation is deeply situated near the root of the lung, the pleura escapes. It is, however, questionable whether lobar pneumonia ever remains central, or whether every lobar pneumonia which has its point of inception near the root of the lung eventually does not progress and involve the periphery of the lung.

Lobar pneumonia, as the name implies, usually involves the major portion of a lobe or an entire lobe of the lung. The disease is in the majority of instances unilateral. In relative frequency the different portions of the lungs are involved in the following order: lower right lobe, lower left lobe, middle right lobe, upper right lobe, upper left lobe, an entire lung, or rarely both lungs.

According to the distribution of the disease several clinical types of lobar pneumonia are recognized.

Apical pneumonia affects only the apex of the lung.

Migratory pneumonia successively involves lobe after lobe of the lung in regular progression.

Double pneumonia involves both lungs simultaneously.

Massive pneumonia is a form of the disease in which, in addition to the alveoli, the bronchial tubes of an entire lobe or lung are plugged with fibrinous exudate.

Central pneumonia is a form in which the disease is situated deeply at the root of the lung, and does not at once involve the peripheral portions.

Lobar pneumonia is attended by very definite changes in the cytology of the blood. The hemoglobin and red cells are involved but slightly in these changes, as the pneumococcus has not apparently the property of producing secondary anemia or destructive changes in the erythrocytes. The predominant change is noted in the leucocytes, which are frequently increased to a remarkable degree. Numerical counts of 12,000, 15,000 or 20,000 are frequently seen; and cases have been recorded in which the count reached 100,000 cells per cubic millimeter. On the other hand, isolated cases are encountered in which the leucocyte count is low, a finding which usually augurs a fatal issue of the disease.

The most characteristic change in the urine during lobar pneumonia is a marked reduction in the elimination of chlorides. Febrile albuminuria is also frequently noted, and occasionally the urine contains elements referable to a complicating acute nephritis.

With the establishment of an extensive area of consolidation in lobar pneumonia, the arterial tension in the lesser circulation is raised, and an additional burden is thrown upon the right ventricle, as evinced by accentuation of the second sound at the pulmonary area during the height of the disease. Frequently, in spite of the increased load, the myocardium maintains its integrity throughout the course of the disease; but in certain instances it becomes the seat of acute myocarditis, with dilatation and abrupt cardiac failure.

Pericarditis occurs in approximately 12 per cent of cases of lobar pneumonia. The disease may assume the acute fibrinous form, or there may be a serous or purulent effusion.

Endocarditis likewise occurs in approximately 10 per cent of the cases. The lesions are commonly limited to the left side of the heart, and the disease may assume the simple or the ulcerative type. As the infection of the cardiac valves is of hematogenous origin, in about 8 per cent of the cases meningitis also develops, a complication which is almost invariably fatal.

Less frequent complications of lobar pneumonia are represented by acute nephritis, arthritis, phlebitis, otitis media, peritonitis, and general septicemia.

While lobar pneumonia in favorable cases terminates in resolution, with removal of the débris of the consolidation, the disease may terminate by sclerosis, resulting in chronic interstitial pneumonia, while in other instances the morbid process goes on to the formation of pulmonary abscess or gangrene.

Physical Signs.—*Inspection.*—The decubitus of the patient is often suggestive in lobar pneumonia. He may be found lying upon the diseased side or may be found sitting up in bed with the spine curved toward the side of the disease. Herpes labialis is a very common finding in lobar pneumonia; and a red spot or flush upon the cheek of the side of the disease is not an infrequent sign of the disease. The respirations are short, and are frequently accompanied by an expiratory grunt.

In a case of unilateral pneumonia, and the disease in the vast majority of cases is unilateral, inspection reveals restriction of the excursion of the thorax upon the side of the disease, with exaggerated excursion of the sound side. The diseased side does not expand to its normal physiologic capacity for two reasons: the air space in the lung is actually decreased by consolidation; and, moreover, the pleurisy accompanying the pneumonic process causes the patient to inhibit the respiratory movements as much as possible. The sound lung, on the contrary, expands vicariously in the effort to compensate as far as may be for the deficiency of expansion of the diseased lung. Litten's diaphragmatic shadow is abolished upon the affected side of the thorax when the lower lobes are involved.

In cases in which the left lung is involved anteriorly, the cardiac impulse is frequently unduly prominent and extensive, as the portion of this lung which overlaps the heart is enlarged and more or less firm and is pushed forward before the heart with each impact of that organ during ventricular systole.

Palpation.—Upon palpation of the thorax over the area of consolidation in the stage of red hepatization vocal fremitus is markedly exaggerated. This increase of fremitus over the base posteriorly, where it is usually encountered in lobar pneumonia, is very striking since under normal conditions of the lung, the fremitus is very faint in this region. The fremitus presents little alteration during the period of engorgement; it reaches its maximum intensity during the stage of red hepatization; and it gradually resumes the normal intensity with the supervention of resolution.

There are two conditions under which vocal fremitus is entirely abolished over an area of consolidation in lobar pneumonia. If the main bronchus leading into this area becomes plugged with fibrinous exudate, as is frequently the case in massive pneumonia, the vocal vibrations are not appreciable to the palpating hand. Again, if there be extensive involvement of the pleura with considerable effusion, the fluid effectually masks the otherwise palpable vibrations during phonation.

During the period of engorgement and the early days of hepatization, the pulse is full and bounding, as the heart reacts powerfully to the constitutional toxemia and the relatively high pressure in the pulmonary circulation. During later stages of the disease the myocardium is liable to a variable degree of parenchymatous change, whereupon the pulse becomes more rapid, sometimes small, and frequently arrhythmic. As a result of the undue acceleration of respiration which is characteristic of lobar pneumonia, the pulse-respiration ratio is markedly disturbed. Instead of presenting the normal ration of 4.5 to 1, a ratio of 3 to 1, 2 to 1, or occasionally even 1 to 1 may be observed.

Blood pressure readings in lobar pneumonia are largely governed by the degree of constitutional toxemia and the state of the myocardium. As pointed out by Gibson, as long as the systolic blood pressure measured in millimeters of mercury does not fall below the pulse rate expressed in beats per minute, the patient is not in immediate danger of cardiac failure. On the other hand, a fatal issue is preceded by a marked fall in blood pressure, occurring coincidentally with myocardial degeneration.

Percussion.—During the period of engorgement, during the first twenty-four hours of the disease, percussion yields either normal resonance or a moderately hyperresonant note. With the establishment of extensive consolidation, involving a considerable portion of a lobe of the lung, percussion yields dullness or frank flatness over the distribution of the disease, unless the consolidation be superimposed upon a large bronchus, whereupon the tympany of the latter mingles to a slight degree with the dullness produced by the area of airless tissue. Light percussion immediately above the level of the consolidation, on the contrary, yields a moderately hyperresonant note, Skodaic resonance, due to relaxation of the tissues which are compressed by the adjacent consolidation.

In a case of central pneumonia, in which the area of consolidation is covered by a rather thick bed of crepitant pulmonary tissue, percussion of the thorax yields normal vesicular resonance, or, in certain cases, Skodaic resonance due to relaxation of the peripheral portion of the lung. As pointed out in a previous section, deep percussion seldom may be expected to elicit impairment of resonance in central pneumonia.

During the latter portion of the stage of gray hepatization and during the stage of resolution, with the gradual breaking down of the consolidation, percussion yields only impaired resonance or moderate dullness over the area of disease.

The elicitation of flatness over the base, in a patient presenting the clinical symptoms of lobar pneumonia, commonly points to the presence of serous or purulent effusion in the corresponding pleural cavity. Here, again, ascending percussion usually reveals the presence of Skodaic resonance immediately above the level of the effusion. Frequently a similar note is elicited over the anterior surface of the thorax in the infraclavicular region upon the corresponding side.

Auscultation.—During the period of engorgement the breath sounds are quiet and partially suppressed, while at the completion of full inspiration there occurs a very valuable physical sign; namely, a fine crepitant râle, the crepitus indux. This râle is produced by the separation of the walls of the infundibula, which have become adherent by tenacious secretion, and it presents to the ear of the examiner a shower of fine crackling sounds.

When the consolidation is fully established, in the stage of red hepatization, the crepitus indux is replaced by distinct bronchial or tubular breathing. During the latter portion of the stage of gray hepatization and during the period of resolution, when the consolidation becomes macerated and partially dissolved, the bronchial breath sounds are replaced by bronchovesicular breathing and subcrepitant râles, constituting the crepitus redux of this disease.

Over the distribution of a dense consolidation vocal resonance is exaggerated, and the voice sometimes possesses a peculiar reverberating quality somewhat similar to the egophony elicited above a pleural effusion. This alteration of the vocal sounds may often be noted immediately above the level of the consolidation when absent over the densest portion of the pulmonary disease. Occasionally the inarticulate whisper is transmitted to the ear even low down over the base of the lung in the presence of an area of dense consolidation, which is traversed by a bronchus with patent lumen.

A pleural friction sound is occasionally detected over the site of the lesion, intermingled with the bronchial or bronchovesicular respiration and râles of the pneumonic process. The establishment of effusion is attended by a gradual masking of the physical signs of consolidation upon auscultation, and the appearance of the cardinal signs of pleurisy with effusion.

Auscultation commonly reveals an accentuation of the second sound at the pulmonary area, occurring as a sign of the increased burden which is thrown upon the right ventricle as a result of the obstacle to the pulmonary circulation occasioned by the consolidation. Similarly, there is occasionally to be detected a reduplication of the second sound at the base, which is due to asynchronous closure of the aortic and pulmonary valves.

In the presence of degenerative changes in the myocardium, the heart sounds at the apex are apt to become high-pitched and valvular, and disturbances in cardiac rhythm may be in evidence. Careful daily auscultation of the precordia should be practiced for the purpose of detecting a complicating pericarditis or endocarditis. The onset of the latter disease is commonly indicated by the appearance of an impure first sound or a soft systolic murmur over the mitral area.

X-ray films during the early stage of the disease, with imperfect consolidation, show a diminution of the transparency of the pulmonary tissues in the area of the disease, usually having its inception around the bronchi, more rarely peripherally. When consolidation is fully established, a dense shadow corresponding to the lobe or lobes involved is noted upon the film. This shadow may be sufficiently dense to obscure the shadows cast by the ribs which overlap the area of consolidation.

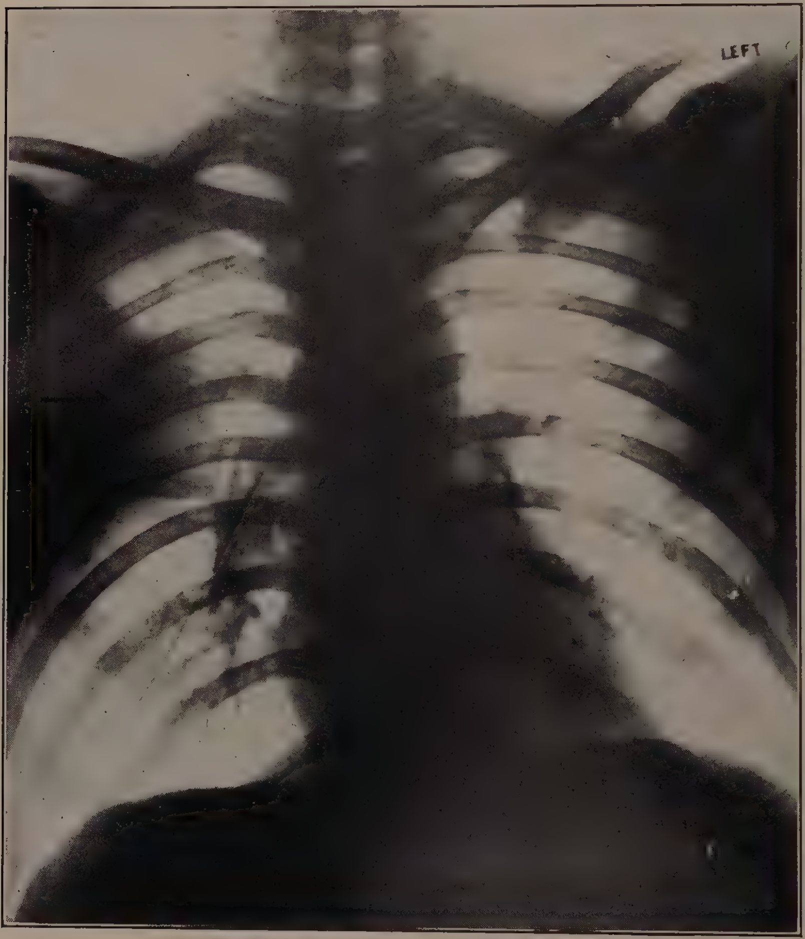

Fig. 281.—Radiograph of lobar pneumonia. Arrow indicates area of consolidation.

Diagnosis.—Uncomplicated lobar pneumonia in an adult, characterized by abrupt onset with pain in the side, initial chill followed by a rapid rise of fever, acceleration of the pulse beyond the degree of pyrexia, herpes labialis, leucocytosis and more or less characteristic sputum, is diagnosed with little difficulty. But in infants, in the aged, in alcoholic subjects, and in the case of a terminal or secondary pneumonia which complicates other conditions such as carcinoma, nephritis or diabetes, a diagnosis may be possible only after considerable observation of the patient. Under these circumstances special significance attaches to deficient expansion of one side of the thorax, fever, acceleration of the pulse, exaggeration of vocal fremitus upon palpation, dullness or flatness upon percussion, and bronchovesicular or bronchial breathing attended by moist râles upon auscultation. In this connection it should be remembered that in massive pneumonia vocal fremitus is apt to be diminished or abolished over the area of disease as a result of occlusion of the bronchi supplying the part. Moreover, dullness is sometimes preceded and is apt to be followed by a moderately hyperresonant note, occurring prior to and following complete hepatization. So also are breath sounds absent or atypical in the presence of incomplete hepatization of the lung, in partial occlusion of the bronchi, and in the presence of a complicating pleural effusion.

The fever in lobar pneumonia usually terminates by crisis in from five to ten days.

The localization of the disease in the lung is sometimes prone to influence the physical signs and to occasion difficulties in diagnosis. In apical pneumonia the signs are those of acute pneumonic phthisis, and the onset of the two diseases is similar. Central pneumonia fails to give the typical signs of lobar pneumonia. In this instance, the lesion, starting deeply within the lung, yields at best a bronchovesicular respiratory murmur, while pulmonary resonance is seldom impaired to an appreciable degree. Forcible percussion under these circumstances seldom succeeds in eliciting dullness, owing to the intervention of a relatively thick bed of crepitant pulmonary tissue.

Lobar pneumonia is not infrequently a source of pain in the right lower quadrant of the abdomen, simulating in this respect acute appendicitis, or in the right upper quadrant, simulating gall bladder disease. In other cases the disease is ushered in with constipation, abdominal pain and meteorism, suggesting intestinal obstruction. Again, in alcoholic subjects cerebral symptoms are apt to predominate the clinical picture, while in children there may be convulsions, and rusty sputum is frequently absent throughout the course of the disease.

Differential Diagnosis.—Lobar pneumonia must be differentiated from acute pneumonic phthisis, bronchopneumonia, pulmonary infarction, pulmonary edema, pulmonary congestion from cardiac defects, and from pleurisy with effusion.

Acute Pneumonic Phthisis.—In the early stages of the disease it is often impossible to differentiate between this disease and lobar pneumonia. This form of pulmonary tuberculosis begins abruptly with chill, pain in the side, and cough which is attended by sputum which is at first mucoid, and later rusty. The physical signs are those of consolidation of one or more lobes, or possibly of an entire lung. The chill is followed by a rapid rise of fever, and the picture is for a time that of frank lobar pneumonia. But at the seventh or ninth day no crisis occurs; on the contrary, the fever persists, and is attended by night sweats, while elastic fibers and tubercle bacilli appear in the sputum. Evidences of softening soon become manifest, with moist or gurgling râles and signs of cavitation.

Bronchopneumonia.—Bronchopneumonia is usually gradual in onset and is usually secondary to other infectious fevers, as measles, influenza, scarlatina, or typhoid fever; or it occurs as the result of the aspiration of material from disease of the upper respiratory passages, or from the decomposition of the contents of bronchiectatic dilatations.

The distribution of the disease is of assistance in differentiation, bronchopneumonia affecting small areas bilaterally, whereas lobar pneumonia, as a rule, affects one lobe or one lung. Bronchopneumonia has been considered as a disease of young children; but especially in connection with influenza, it is very commonly seen in young and middle-aged adults.

The physical signs of bronchopneumonia are diffuse, the patches of consolidation being scattered widely over both lungs, so that the percussion note is never frankly dull or flat, but there is bilateral impairment of pulmonary resonance of moderate degree. Similarly, bronchopneumonia is not attended by frank bronchial breathing, but by bronchovesicular breath sounds. Crisis does not occur in bronchopneumonia, the fever resolving by lysis.

Pulmonary Infarction.—Infarction of the lung is abrupt in onset, with dyspnea and cough; and the expectoration is not viscid, but is fluid and often tinged with blood or the disease is attended by frank hemoptysis. In simple infarction the febrile movement does not equal that of lobar pneumonia; but in septic infarction a local pneumonia frequently develops at the site of the infarct, and results in pulmonary abscess with its hectic temperature and colliquative sweats. There are often signs of an associated valvular heart lesion, as the disease often occurs in persons with chronic endocarditis.

Unless they are very extensive, pulmonary infarcts produce very few definite physical signs, though extensive infarction of the lower lobe of the lung produces bronchovesicular or bronchial breathing when properly situated with reference to a large bronchus.

Pulmonary edema develops commonly in patients with valvular heart lesions or nephritis, producing extreme dyspnea or orthopnea, with numerous moist or bubbling râles over the bases posteriorly. The respiratory sounds are diminished in intensity rather than purely bronchial. The sputum is abundant and characteristic; and the disease in its manifestations is bilateral, affecting the bases of both lungs, and it is not attended by fever.

Pulmonary Congestion.—The variety of pulmonary congestion which most closely simulates lobar pneumonia is the acute active congestion which is described by the French authors as Woillez's disease, a form of active congestion of sudden onset, which really constitutes a larval type of pneumonia. Hypostatic congestion is bilateral in its manifestations, with moist râles at the bases of the lungs; but there is no fever, and the sputum is not viscid or rusty.

Serofibrinous pleurisy with effusion may be confused with lobar pneumonia; but there are sufficient diagnostic points to differentiate the two diseases with comparative ease.

In pleurisy with effusion the onset is gradual, with chilly sensations rather than abrupt with a distinct chill as in the case of lobar pneumonia. Pleurisy in the early stage is almost always accompanied by a pleural friction sound, which disappears as the effusion accumulates in the pleural cavity. Pleuritic fever resolves by lysis, while the fever of lobar pneumonia terminates by crisis. In pleurisy with effusion there is often a tuberculous history obtainable in connection with the pleurisy. In pleurisy with effusion vocal fremitus is abolished, instead of exaggerated as is the case in lobar pneumonia. There is flatness upon percussion, whereas in pneumonia the note is dull or flat, but is preceded and followed by a hyperresonant note during the stages of incomplete hepatization. Instead of the crepitant and subcrepitant râles of pneumonia, in pleurisy with effusion there is total abolition of respiratory sounds as a rule over the effusion. Aspiration of the thorax reveals fluid in the case of pleurisy with effusion.

Herpes labialis is rare in pleurisy, and very common in lobar pneumonia. Egophony may be elicited below the scapular angle with fair constancy in pleurisy with effusion; and the cardiac apex beat is displaced to the side opposite to the effusion. The intercostal spaces are more apt to be obliterated or to bulge in pleurisy with effusion than in pneumonia. Visceral displacement is always more pronounced in pleurisy than is the case with lobar pneumonia.

ATYPICAL PNEUMONIAS

Not only during the epidemic of influenza, but frequently in the average practice or hospital service, an intermediate type of pneumonia is noted. Such an atypical pneumonia has some of the characteristics of both lobar and bronchopneumonia.

Clinical Pathology.—It usually follows an acute inflammation of the throat and upper air passages. The consolidation most frequently occurs in the lower portion of the left upper or left lower lobe or in the right middle or right lower lobe. The size of the consolidation varies from the size of a walnut to the size of the palm of the hand. It is dense in the center and fades at the edges into normal lung structure. Young and middle-aged adults seem most susceptible but it may occur at any age.

No organism has been isolated which appears to be pathogenic.

The symptoms are general malaise, cough with a production of a rather thick pinkish or bloody sputum. This sputum is less viscid than the sputum of lobar pneumonia. The fever rises slowly, reaching a maximum of 102 or 103 degrees about the second day, remains constant for from three to seven days, and falls slowly, reaching normal in from nine to twelve days. Resolution is usually accompanied by fibrosis and thickening of the pleura, which rarely completely disappears.

The urine usually contains considerable albumin and granular casts. The leucocyte count is frequently between 4000 and 7000, but cases with a leucocyte count of 20,000 are occasionally met with.

The Physical Signs.—*Inspection.*—The face is often flushed, cough is not so distressing as in lobar pneumonia, and the sputum is raised with less difficulty. Slight diminution of expansion is noted when the consolidation is of considerable size and Litten's sign is usually shortened on the affected side.

Palpation reveals increase of vocal fremitus over the area of consolidation. The pulse is usually comparatively slow, being rarely above 90 per minute when the temperature is 102.

Percussion demonstrates an area of dullness which frequently increases in extent for a day or two, remains stationary for four to seven days, then gradually diminishes. Diminished resonance, however, may be noted for months and even years due to fibrosis and thickened pleura. The dullness is most marked in the center and gradually fades with normal resonance.

Auscultation.—Over the area of consolidation bronchial breath sounds accompanied by medium-sized moist râles are noted during the acute stage, the râles gradually disappearing and the breath sounds becoming more distinct and of a bronchovesicular character as resolution proceeds. Spoken and whispered voices are accentuated. Occasionally voice and breath sounds are absent, due to bronchial plugging.

The heart sounds are usually slow and strong, with variable accentuation of the pulmonary second sound.

The X-ray.—During the height of the disease a shadow more dense in the center, gradually fading at the edges, corresponds to the area of percussion dullness. After resolution there usually remains some haziness due to fibrosis of the lung and thickened pleura. Films taken two years after recovery still show evidence of fibrosis.

Diagnosis.—This form of pneumonia is preceded by red throat and symptoms of upper respiratory inflammation. The consolidation is unilateral and of considerable size, but rarely if ever involves an entire lobe. The consolidation is rarely noted in the upper portion of the upper lobes. The symptoms appear and disappear gradually. The pulse is slow and the leucocyte count is usually below 8000. The sputum is frequently bloody or pinkish and is moderately viscid.

Differential Diagnosis.—*From acute bronchitis* by the general severity of symptoms and the bloody viscid sputum and the demonstrable area of consolidation.

From Lobar Pneumonia.—The onset is gradual, rarely accompanied by a chill, herpes though occasionally seen are not so common, the sputum is more bloody than prune juice, the area of consolidation is smaller and is rarely surrounded by skodaic resonance. The pulse is usually slow, the temperature falls by lysis. Fibrosis is more common.

Bronchopneumonia.—The preceding throat symptoms are more common, the disease is unilateral, with a single area of consolidation, the pulse is strong, and the leucocyte count not so high.

Acute Bronchopneumonic Phthisis.—The history and previous health of the individual may be suggestive.

The rarity of apical involvement and the bloody or pink sputum may be present in tubercular bronchopneumonia, but usually takes the form of hemoptysis. The presence of tubercle bacilli in the sputum will clinch the diagnosis.

The size and location of the consolidation may be the same but bilateral signs are more likely to occur in tubercular bronchopneumonia.

The pulse is usually more rapid and the leucocyte count higher in tubercular pneumonia.

X-ray examination usually shows evidence of old tubercular lesions.

BRONCHOPNEUMONIA (LOBULAR OR CATARRHAL PNEUMONIA)

Clinical Pathology.—Bronchopneumonia, catarrhal, or lobular pneumonia is an acute inflammation of the terminal bronchioles, spreading secondarily to the adjacent alveoli, which become filled with inflammatory exudate.

Bronchopneumonia is almost invariably a secondary disease, following many of the acute infectious fevers, notably pertussis, measles, and influenza, diseases which during their course are associated with a variable degree of acute bronchitis. In these cases the development of bronchopneumonia represents a downward extension of the bronchitis to the finer bronchioles, where it constitutes a capillary bronchitis.

Bronchopneumonia also follows the aspiration into the bronchioles of food particles from the buccal cavity, or of secretions and blood from the upper respiratory passages, constituting deglutition or aspiration pneumonia. During operative procedures upon the nose and throat, in apoplexy, and during comatose states from other causes, infectious material may be aspirated into the bronchi and induce bronchopneumonia. Deglutition pneumonia occurring in the newly born is due to the aspiration of secretions from the birth canal during parturition.

Bronchopneumonia is especially prone to attack infants and children under two years of age. A primary bronchopneumonia not infrequently develops in these subjects, with abrupt onset simulating closely that of lobar pneumonia.

In adults, on the contrary, bronchopneumonia is less frequent than is the lobar form of pneumonia. Many of these cases follow affections of the respiratory organs, notably influenza; but any general or focal infection may initiate a catarrhal pneumonia under favorable circumstances. Again, bronchopneumonia is a frequent terminal affection in elderly subjects who are debilitated by nephritis, carcinoma or diabetes.

As bronchopneumonia is usually a secondary disease, so also is it commonly induced by mixed infection. Various strains of streptococci are the predominant organisms recovered in the majority of cases. In other instances the disease is caused by the influenza bacillus, the pneumococcus, principally Type IV of this organism, Friedländer's bacillus, or certain strains of staphylococci. In certain epidemics of plague a very fatal type of bronchopneumonia is caused by infection with Bacillus pestis.

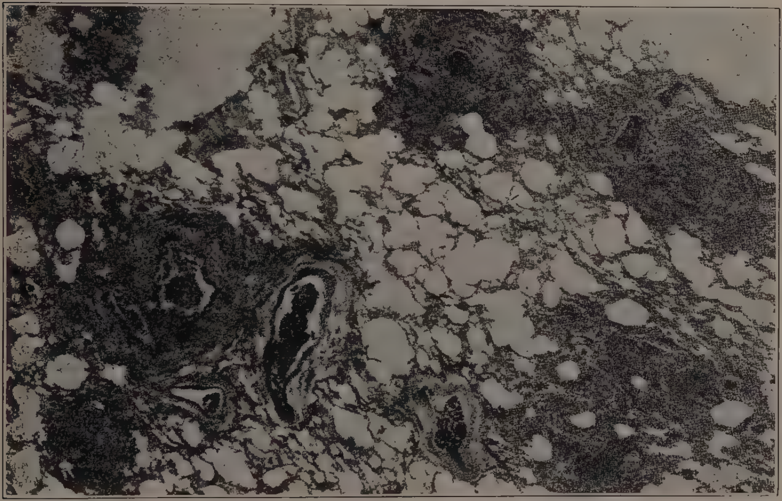

Fig. 282.—Consolidation of bronchopneumonia. (From Delafield and Prudden.)

The symptoms and course of the disease present a certain variety, depending upon the type of the infecting organism. It is known that many cases of streptococcic bronchopneumonia are attended by general septicemia, while the catarrhal pneumonia induced by the influenza bacillus is frequently followed by a variable degree of pulmonary fibrosis. Certain cases run a fulminating course, and terminate fatally in forty-eight to seventy-two hours, while others terminate favorably in two to three weeks. In the presence of complications the illness is frequently greatly protracted.

The inflammatory changes in bronchopneumonia have their inception in the smaller bronchi and terminal bronchioles, whence the disease extends to the communicating pulmonary alveoli, involving the lobules supplied by the infected air passages. The lumen of the bronchiole in the area of the disease is filled with mucopurulent exudate, while the peribronchial tissues are consolidated in the immediate vicinity of the tube, and exhibit splenization for a variable distance beyond the area of consolidation.

Upon section, the pneumonic patches are observed to be situated radially around the communicating bronchus, usually invading the pulmonary tissue for a distance of three to five millimeters. The lesions are largely confined to the peripheral portions of the lungs. The consolidated areas vary greatly in dimension, ranging from points the size of a pinhead to lesions several centimeters in diameter. These areas are commonly dark red, and are slightly elevated above the surrounding surface of the section. Here and there a bronchus occluded by mucopurulent exudate may be seen, and there are frequently small areas of focal suppuration. Areas of atelectasis are common, particularly along the margins of the lungs, occurring as a result of complete obstruction of the communicating bronchioles.

Microscopically, the bronchiolar walls are thickened, with desquamation of the lining cells, while the lumen contains an exudate composed of desquamated bronchiolar cells, bacteria, leucocytes, and a minimal number of erythrocytes.

The pleura is usually involved in the inflammation, exhibiting lusterless areas, which correspond to the distribution of the patches of peripheral consolidation. In cases of the pseudolobar type acute plastic pleurisy occasionally is noted, or there may be free fluid in the pleural sac. The effusion of catarrhal pneumonia is very prone to assume the purulent form.

Bronchopneumonia is in the vast majority of cases bilateral, attacking both lungs simultaneously, although the pathologic changes are seldom equal upon the two sides. As the designation "lobular" implies, the disease exhibits a tendency to involve lobules rather than lobes of the lung. As a general rule, this principle holds true in the distribution of the lesions, which usually conforms to one of two types. Thus, in the disseminated form of bronchopneumonia, lobules are consolidated widely throughout the lung; but the areas of consolidation are small, and are separated by bands of crepitant pulmonary tissue, which are the seat of compensatory emphysema. In the pseudolobar form of the disease, on the contrary, a number of contiguous lobules are consolidated, occasionally affecting the greater portion of a lobe of the lung. This distribution of the lesions is noted especially in acute streptococcic bronchopneumonia, in which the distribution of the disease and the physical signs are very suggestive of lobar pneumonia.

The most frequent complication of catarrhal pneumonia is pleurisy, acute fibrinous or serofibrinous in type. Effusions complicating this disease are very prone to assume the form of empyema. Otitis media is not an infrequent complication in young subjects. Endocarditis, pericarditis, and meningitis occasionally occur as a result of hematogenous infection.

The disease is not attended by striking alterations in the cytology of the blood, save in cases complicated by empyema, in which secondary anemia frequently attains an extreme degree. In robust subjects the disease is attended by moderate leucocytosis.

The urine of bronchopneumonia patients is usually highly colored, occasionally contains a trace of albumin, and, with less frequency, tube casts due to an acute nephritis.

Catarrhal pneumonia usually terminates by resolution, with liquefaction of the cellular exudate, and a gradual return of the diseased lungs to a normal state. Occasionally, however, particularly in cases due to infection with the influenza bacillus, the disease terminates in disseminated or insular sclerosis of the lungs. Again, in the case of deglutition or aspiration pneumonia, due to the aspiration into the bronchi of infectious material, the disease sometimes terminates in abscess or gangrene of the lung.

Physical Signs.—*Inspection.*—In infants and young children bronchopneumonia is usually attended by signs of stenosis of the bronchioles, as evinced by inspiratory dyspnea, in which the accessory muscles of respiration are called into play, together with inspiratory retraction of the lower intercostal spaces and epigastrium. In this class of cases the disease is sometimes ushered in with convulsions. Moderate cyanosis of the mucous membranes is common, and suffocative attacks are frequent.

In the established case of bronchopneumonia in an adult the respirations are accelerated, and the thoracic expansion is bilaterally restricted. Moreover, in instances of extensive consolidation of the lower lobes, the upper portion of the thorax exhibits a greater degree of expansion than does the lower portion. In cases attended by extensive atelectasis of the anterior border of the left lung, which overlaps the heart, an extended area of cardiac impulse may be visible, whereas in other cases compensatory emphysema of this border serves to obscure the impulse of the heart.

Cough is commonly distressing, and is attended by a variable quantity of expectoration, which is moderately tenacious and glairy, sometimes sanguineous, but containing no elements which are pathognomonic of the disease.

Palpation.—Light finger-tip palpation of the lower lateral thoracic regions sometimes reveals the presence of a minor degree of lagging of the bases, which may have escaped detection upon inspection of the thorax. In a subject with a thin chest wall, palpation likewise usually elicits a moderate exaggeration of vocal fremitus when there is an area of confluent consolidation of sufficient dimensions, which is favorably situated with reference to the periphery of the lung and one or more of the larger bronchi. Indeed, in the pseudolobar form of the disease, a moderate exaggeration of tactile fremitus is the rule, whereas in the disseminated form the intervening emphysematous portions of the lungs effectually mask any increase of fremitus which is produced by the small areas of peribronchial consolidation. Similarly, partial occlusion of the bronchi by inflammatory exudate is attended by diminution or absence of fremitus over an area corresponding to the distribution of the occluded air passages.

During the early days of a bronchopneumonia, the pulse exhibits a striking acceleration, with a relatively large volume and usually a regular rhythm. The tension of the pulse and the resistance to manual compression vary in individual cases, depending upon the age and physical state of the patient. In children the pulse is commonly readily compressed, whereas in terminal cases of bronchopneumonia, which complicate arteriosclerosis or nephritis in elderly patients, the pulse is usually of high tension and is compressed with effort. In the latter class of patients various disturbances of rhythm are apt to be encountered during the course of the disease. In cases presenting an unfavorable prognosis, in all classes of patients, the tension of the pulse falls, while the rate becomes more rapid, and the pulse is frequently arrhythmic.

Percussion.—In bronchopneumonia during the first twenty-four hours of the disease, percussion of the thorax elicits only normal pulmonary resonance; but in a suspected case the bases posteriorly should be percussed daily; and in most instances at the expiration of forty-eight hours one or more areas of dullness will have become demonstrable in these regions. However, the thorax of the young child, in whom the disease is frequently encountered, is normally slightly hyperresonant; and, moreover, the areas of compensatory emphysema, which surround and separate the patches of consolidation, tend to mask any dullness which would be produced by the presence of the consolidated areas, and to impart a vesiculotympanitic quality to the percussion note. In the disseminated form of bronchopneumonia frank dullness is not to be expected, as the hyperresonance of the associated compensatory emphysema effectually masks the dullness of the consolidation. In the pseudolobar form, on the contrary, dullness may always be elicited by careful daily percussion of the bases posteriorly. In demonstrating the dullness of superficial patches of consolidation it is essential to employ very light superficial percussion.

Percussion of the anterior portion of the thorax in the infraclavicular and mammary regions yields Skodaic resonance. The presence of bilateral dullness over the bases posteriorly with Skodaic resonance anteriorly is very suggestive of the consolidation of bronchopneumonia.

Auscultation.—Auscultation of the bases posteriorly reveals upon consecutive examinations the downward extension of the original bronchitis. In addition to subcrepitant râles incident to this disease, crepitant râles become audible upon the completion of inspiration, indicating the participation of the pulmonary alveoli in the inflammatory process. At times the râles are partially obscured by plugging of the bronchioles by inflammatory exudate; but inspiration following expiration and cough usually serves to bring them again into evidence.

With the formation of numerous patches of consolidation, the respiratory sounds assume a bronchovesicular character, and, in the presence of a dense pseudolobar consolidation, true bronchial respiration is to be elicited over a base posteriorly. In the latter class of cases it is occasionally possible to elicit bronchophony or pectoriloquy over an extensive peripheral consolidation, which extends inward toward one of the larger bronchi.

During the height of the disease the second sound of the heart at the pulmonary area is commonly accentuated, as a result of the increased arterial pressure in the pulmonary circuit. As long as the myocardium maintains its integrity, the first sound at the apex is booming and muscular. A change in the quality of the first sound in the direction of a high-pitched valvular sound of brief duration, is suggestive of myocardial degeneration. A complicating endocarditis is ushered in by the appearance of an impure first sound or a systolic murmur at the mitral area.

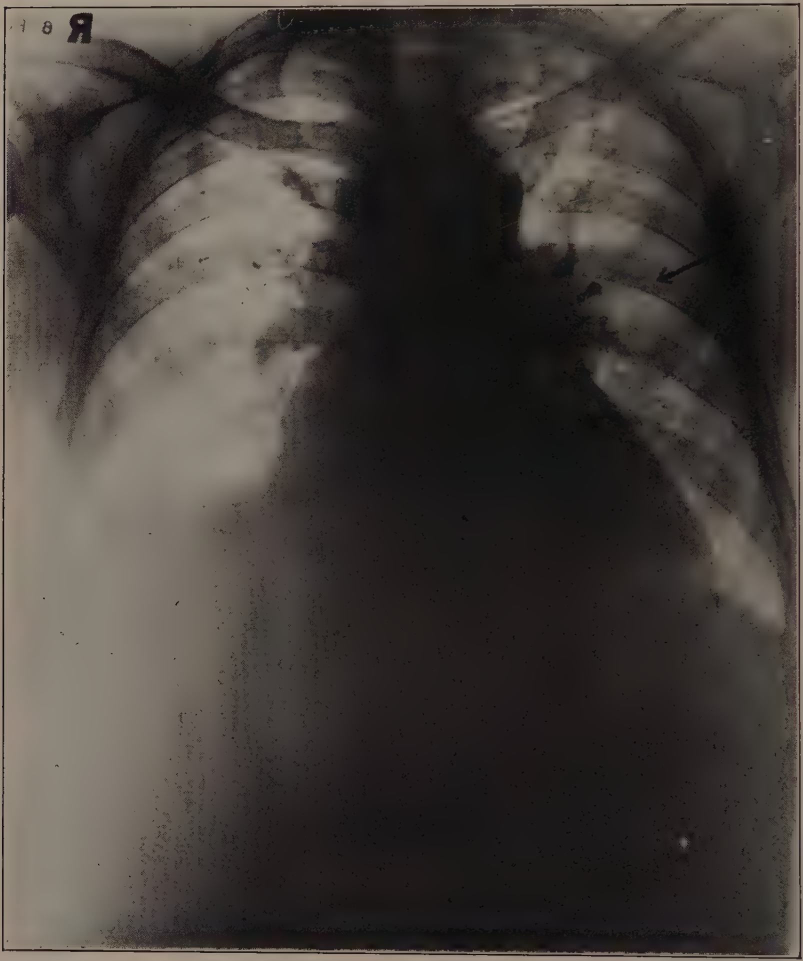

Fig. 283.—Radiograph of bronchopneumonia. Arrow indicates small areas of consolidation.

X-ray.—The x-ray film shows bronchial markings more pronounced, with scattered areas of density of varying size, usually noticeable in both lungs.

Diagnosis.—While the physical signs of bronchopneumonia are bilateral in their distribution, they are seldom present to the same degree upon the two sides of the thorax, as one lung is usually involved to a greater extent than is the other. In all cases in which physical signs of a capillary bronchitis are elicited over one lung, a careful search for similar signs over the opposite lung should be made.

Frequently a diagnosis of catarrhal pneumonia cannot be made upon the physical signs alone, as these are often meager and misleading, and as the mode of onset of the disease is not infrequently atypical. The physical signs should be construed in conjunction with the fever, cough, inspiratory dyspnea, cyanosis, and pain in the thorax, arising during an attack of acute bronchitis or one of the acute infections. Not infrequently the picture of bronchopneumonia is overshadowed for a time by the symptoms and signs of the original disease. The bilateral distribution of the signs in the bases of the lungs, which obtains in the majority of the cases, should be sufficient to suggest the presence of bronchopneumonia.

Differential Diagnosis.—From *acute bronchitis,* bronchopneumonia is differentiated by the absence of areas of hyperresonance and impaired resonance in the former, and by the finer quality of the râles in the latter disease. Moreover, in acute bronchitis there is little or no fever, and the disease is altogether milder in its clinical manifestations.

From *lobar pneumonia,* bronchopneumonia presents many points of differentiation. Bronchopneumonia is usually secondary to another disease and is of insidious onset; whereas lobar pneumonia is usually a primary disease, with abrupt onset and stormy course. Moreover, bronchopneumonia is a bilateral pulmonary disease, producing multiple areas of impaired resonance over both lungs, rather than a single area of dullness or flatness over one or more lobes of a lung. Also, the fever of bronchopneumonia terminates gradually by lysis, whereas that of lobar pneumonia terminates by crisis between the seventh and the ninth days in the majority of cases. As a rule, bronchopneumonia attacks subjects of impaired vigor who are below par physically, whereas lobar pneumonia frequently attacks robust persons in the prime of health.

In the rarely encountered primary form of bronchopneumonia, and in those cases in which the disease assumes the pseudolobar form, in which the pneumonic patches fuse and involve the greater portion of a lobe of the lung, the differentiation from lobar pneumonia is attended with considerable difficulty. In this class of cases the physical signs are very similar if not identical; but in bronchopneumonia there is almost invariably apparent, even if to a slight degree, some involvement of the opposite lung. Moreover, in lobar pneumonia the sputum is fairly characteristic, differing markedly from the viscid, glairy expectoration of bronchopneumonia.

From *acute bronchopneumonic phthisis,* bronchopneumonia cannot be differentiated in the incipient stage. Involvement of the apices by the disease is suggestive of phthisis; but in certain cases of bronchopneumonic phthisis the apical involvement is not particularly prominent. However, in phthisis the temperature is prone to remain more uniformly high, and to be interrupted early in the course of the disease by night sweats. There is usually obtainable a history of intimate association with a tuberculous person or evidence of a tuberculous focus at some point. Moreover, in acute bronchopneumonic phthisis emaciation progresses rapidly, the course of the disease being progressively downward, until eventually tubercle bacilli are demonstrable in the sputum.

Bronchopneumonia in infancy is sometimes with difficulty differentiated from meningitis, as the disease in this class of patients is often accompanied by marked cerebral symptoms, as delirium or convulsions. Examination of the spinal fluid serves to clear up the diagnosis in these cases.

CHRONIC INTERSTITIAL PNEUMONIA

(PRODUCTIVE PNEUMONIA; CIRRHOSIS OR FIBROSIS OF THE LUNG)

Clinical Pathology.—Chronic interstitial pneumonia, productive pneumonia, pulmonary cirrhosis or fibrosis, is a chronic indurative pulmonary disease, representing the ultimate consequences of prolonged irritation of the fixed connective tissue elements of the lungs, pursuing an irregular course and attended by progressive emaciation, cough, dyspnea, and more or less characteristic deformity of the thorax. The predominant tissue change of the disease is the formation of an excessive amount of fibrous tissue, which, by subsequent contraction frequently results in bronchiectasis and diminution of the bulk and vital capacity of the lung. The designation "pulmonary fibrosis" is most applicable in the majority of cases, in which no signs of a pneumonic process are demonstrable.

According to the distribution of the lesions, three types of pulmonary fibrosis are recognized; namely, the *local* or *insular* form, the *massive* or *lobar* form, and the *pleurogenous* form of the disease.

The *insular* form of pulmonary fibrosis most frequently involves the upper lobe of one lung, in which the pathologic changes are most pronounced in the region of the apex, suggesting that the provocative agent may have been infection with the tubercle bacillus. However, many of these cases do not exhibit the specific histologic changes which are associated with healed phthisis. It is probable that some of the cases are due to the prolonged inhalation of irritant dusts during an injurious occupation, and that they represent a rudimentary form of pneumonoconiosis.

In other instances the insular sclerosis involves portions of the lower lobes, usually resulting from a previous bronchopneumonia, particularly the form of the disease which is caused by the influenza bacillus. Other cases, again, present the maximum involvement about the hilus regions, in which event chronic bronchitis or pulmonary syphilis may be the underlying cause. Local areas of atelectasis are responsible for certain cases of insular pulmonary fibrosis. In this event, the disease involves chiefly the peripheral portion of the lung.

The principal distribution of the fibrosis is peribronchial in cases of bronchopneumonic origin, and peripheral in those due to atelectasis. In this type of the disease a variable portion of a lobe of the lung is shrunken; the tissue cuts with distinct resistance; and the surface of the section exhibits an extensive destruction of the alveolar structure of the lung, which is replaced by firm bands of fibrous tissue. The destruction of the investing capillary network, by impairing the integrity of the pulmonary circulation, results in dyspnea, which is sometimes extreme. As a result of the traction exerted upon the bronchi by the fibrous bands, ectases of these tubes are frequently encountered. The portions of the lung which surround the areas of fibrosis are in a state of compensatory emphysema.

Over the site of peripheral areas of sclerosis, the pleura is thickened and lusterless; and adhesions between the visceral and the parietal pleura are relatively common findings.

The *massive* or *lobar* form of pulmonary fibrosis is almost invariably a unilateral affection, and the maximum involvement is usually encountered in the lower lobe. In the infrequent cases of bilateral disease, which usually result from chronic passive congestion of the lungs, which terminates in "brown induration" of these structures, the cirrhotic changes are always more pronounced in one lung than in its fellow.

Massive chronic interstitial pneumonia sometimes develops as a result of incomplete resolution after catarrhal pneumonia. The disease in this instance has its inception as a peribronchial fibrosis, which spreads to the pulmonary alveoli along the interalveolar septa, involving various portions of a lobe in the fibrotic process. Not infrequently an entire lobe is invaded; and, indeed, in certain cases an entire lung is involved. Croupous pneumonia is occasionally followed by pulmonary fibrosis, but with less frequency than is the catarrhal form of the disease. The fibrosis resulting from lobar pneumonia commonly begins in the pulmonary alveoli, where the inflammatory exudate undergoes organization, resulting in a condition of "gray induration" of the lung. The distribution of the disease corresponds to that of the original pneumonia, commonly involving a lower lobe of the lung.

Certain cases of massive cirrhosis of the lung follow extensive atelectasis and pulmonary infarction, in which event the distribution of the area of fibrosis is commonly peripheral.

In this form of interstitial pneumonia a large portion of a lobe, an entire lobe, or a lung is diminished in size and its capacity for expansion is restricted to a degree which is commensurate with the extent of the lesion. In extreme cases of massive fibrosis the lung is represented by a small shrunken mass, which occupies the superior portion of the pleural cavity near the vertebral column. Upon section it is observed that dense fibrous bands traverse the pulmonary parenchyma in various directions, resulting in destruction of the alveoli in their path. Bronchiectasis and bronchial stenosis by fibrous bands are frequently encountered.

The investing pleura is thickened; and, over the apical portion of the lung, dense adhesions are sometimes encountered.

Pleurogenous interstitial pneumonia usually develops as a result of compression of the lung by a pleural effusion of extensive duration. In this type of the disease the pulmonary fibrosis has its inception in the pleura, which is excessively thickened. From the pleural membrane dense bands of fibrous tissue penetrate the pulmonary parenchyma in various directions, frequently reaching the larger bronchi in the deeper portions of the lungs. As a result of traction, dilatations of these tubes are frequently encountered.

Adhesions between the visceral and the parietal pleura are usually extensive in this type of the disease, and the pathologic picture in certain cases approaches that of chronic adhesive pleurisy.

In cases of massive interstitial pneumonia of unilateral distribution, the opposite lung is in a state of compensatory emphysema. The diminution of the bulk of the diseased lung permits the mobile diaphragm to ascend into the space formerly occupied by the cirrhotic lung, whereas the muscle is displaced downward upon the opposite side by the compensatorily voluminous sound lung. Similarly, the mediastinum is displaced toward the side of the disease, carrying with it the pericardium and the heart. Such extensive cases of unilateral disease are attended by fairly characteristic deformity of the thorax.

Physical Signs.—As the physical manifestations of pulmonary fibrosis are governed by the distribution and the extent of the lesions which are present in the individual case, it is obvious that there can be no classical picture of the disease, save in the massive form, associated with diminution of the thoracic contents upon one side, with a corresponding increase upon the side of the emphysematous lung.

Thus, cases of fibrosis involving the apical portions of the lungs, afford signs closely simulating those of chronic phthisis, whereas insular sclerosis of the lower lobes is attended by alterations in the respiratory sounds suggesting partial consolidation or collapse of the lung. In such cases a diagnosis cannot be made upon the physical signs alone, which are frequently meager and difficult of interpretation. The massive form of the disease, on the contrary is attended by distinctive physical manifestations which, construed in the light of the subjective symptoms, are of distinct aid in diagnosis.

Inspection.—In advanced cases of massive interstitial pneumonia there is usually to be detected a variable degree of retraction of the affected side, while the shoulder droops. The cardiac impulse is displaced toward the side of the lesion; and, when the disease attacks the left lung, there is usually a wide area of impulse in the third, fourth and fifth interspaces. In cirrhosis of the right lung, on the contrary, the cardiac apex may be displaced so far to the right as to occupy a position behind the sternum, in which event no visible impulse can be made out. Again, even in those cases in which the fibrosis of the right lung is not so great as to lead to displacement of the apex behind the sternum, the impulse may yet be invisible because of overlapping of the heart by the compensatorily emphysematous left lung.

The side of the thorax corresponding to the disease is restricted in its inspiratory excursion; or, in advanced cases, it is immobile. The opposite side of the thorax commonly exhibits an increased excursion as a result of compensatory emphysema of the corresponding lung. The respiration is accelerated, and the patient has difficulty in drawing air to the depths of the lungs.

Palpation.—In massive pulmonary fibrosis palpation serves to confirm the limited excursion of the thorax upon the diseased side, and to accurately localize the cardiac impulse when it is accessible. Vocal fremitus may be exaggerated, impaired, or abolished upon the side of the disease, depending upon the state of the subjacent pulmonary tissues and the thickness of the pleura. Occasionally an exaggeration of tactile fremitus is to be elicited over a large bronchiectasis which approaches the thoracic wall.

Percussion.—In massive interstitial pneumonia percussion of the thorax upon the side of the disease usually yields a dull or flat note; but in certain cases rather forcible percussion of the upper axillary region may yield a moderately tympanitic note, which is caused by the proximity to the thoracic wall of a large bronchiectatic cavity. Percussion of the opposite side of the thorax commonly elicits a hyperresonant note, as a result of compensatory emphysema of the underlying lung.

The superior limit of pulmonary resonance is restricted upon the side of the disease, as a result of retraction of the apex of the lung. In the case of fibrosis of the right lung, retraction of the inferior border causes an extension of hepatic dullness above the level of the sixth rib, whereas in the presence of left-sided disease the tympany of Traube's space extends to an abnormally high level.

Auscultation.—In massive interstitial pneumonia the respiratory murmur is absent over the lower portion of the thorax when the lung is shrunken and occupies the superior portion of the pleural cavity. Auscultation of the upper regions may reveal distant breath sounds, which are apt to have a bronchial or amphoric quality engrafted upon them. Occasionally râles of a coexisting bronchitis may be elicited. In pleurogenous cases respiratory sounds may be absent over the entire side of the disease, simulating in this respect the auscultatory findings of fluid in the pleural cavity. Vocal resonance may be exaggerated or diminished in intensity, depending upon the state of the subjacent pulmonary tissues and the pleura.

The pulmonic second sound is accentuated; and late in the course of the disease is apt to become weakened, the tricuspid systolic "safety-valve" murmur then becoming audible, betokening imminent right heart failure.

X-ray.—The x-ray film usually shows marked increase of hilus shadows, the peribronchial markings are more pronounced, the lung structure is more dense, with bands of fibrous tissue appearing as streaks or stellate markings. The mediastinal space is usually narrowed or obliterated, and the diaphragm shadow is higher on the side most affected.

Diagnosis.—In a subject presenting unilateral retraction of the thorax, with drooping of the corresponding shoulder and other signs of pulmonary collapse of considerable duration, following in the train of pneumonia, pleuritis, or infarction, the massive form of chronic interstitial pneumonia should be suspected. The insular form of the disease is difficult to diagnose with any degree of certainty because of the local distribution of the lesions and the atypical and inconclusive character of the physical signs which they engender.

Again, a slowly growing neoplasm of the lung or pleura for a considerable period closely simulates the physical signs of interstitial pneumonia.

Differential Diagnosis.—It is to be borne in mind that the physical signs of *fibroid phthisis* are very similar in many instances to those of chronic interstitial pneumonia. In phthisis, however, the disease is more frequently bilateral; and the persistence of moist râles at an apex speaks in favor of phthisis. The clinical symptoms and course of the two diseases are so similar that they cannot be utilized in reaching a differential diagnosis. The detection of the tubercle bacillus in the sputum is in the end the deciding factor in the differential diagnosis of the two diseases.

Empyema of extensive duration is not infrequently confused with the massive type of pulmonary fibrosis. In both diseases the pleura is excessively thickened in many instances, and both are prone to present retraction of the affected side, with drooping of the corresponding shoulder, and physical signs which are almost identical. Fluoroscopy of the chest and exploratory puncture are required to make a differential diagnosis in many cases.

Bronchiectasis and chronic interstitial pneumonia are nearly always concomitant diseases. The task in this instance is to determine which morbid pulmonary change predominates in the given case. Marked clubbing of the fingers speaks strongly in favor of bronchiectasis, as does also the raising of large quantities of foul expectoration amid paroxysms of distressing cough.

ACUTE PNEUMONIC PHTHISIS

Clinical Pathology.—Acute pneumonic phthisis, ''phthisis florida,'' or ''galloping consumption,'' is a rapidly spreading caseous pneumonia, caused by infection with the tubercle bacillus, involving the greater portion of a lobe, and occasionally an entire lung. The disease almost invariably attacks an upper lobe of the lung, having its point of inception in an old tuberculous focus in one apex. Why pulmonary tuberculosis assumes this acute exudative form in certain cases has not been adequately explained.

Males are attacked by this form of phthisis more frequently than are females, and the subject of the disease is usually in apparently robust physical condition at the time of the onset of the acute symptoms of the disease. Exposure to inclement weather is not infrequently the immediate precursor to the onset of the disease, while in other instances it develops during convalescence from an attack of influenza or other acute infectious disease.

During the early stage of the disease, corresponding to the stage of dense consolidation, the affected lung is increased in weight, and sinks when placed in water. Upon section, more or less extensive caseous areas are encountered, which present a smooth surface, are relatively dry, airless, and gray or yellowish-white in color. In other instances the surface of the section presents a mottled appearance, which is due to the alternation of areas of caseation and hyperemic patches of consolidation. In late stages of the disease one or more cavities may be encountered.

Microscopically, the pulmonary alveoli are filled with a dense consolidation, exhibiting numerous areas of caseation; and an old tuberculous focus can usually be demonstrated in the consolidated area. Frequently minute cavities are to be noted in the midst of an area of caseation. There is little formation of connective tissue, and no demonstrable effort to limit the spread of the disease by sclerosis.

With the further progress of the disease, necrosis in the interior of the consolidated area results in the formation of more or less extensive cavities. However, a fatal issue may occur before softening and excavation of the lung supervene.

The bronchial lymph nodes are commonly enlarged and occasionally are caseous. Such glands may contract adhesions with neighboring blood vessels, and by rupture into the blood stream result in general miliary tuberculosis.

The pleura corresponding to the distribution of the consolidation is clothed with a variable amount of fibrinous exudate; but pleural effusion and adhesions are seldom found at autopsy.

The onset of the disease is abrupt, simulating that of lobar pneumonia. As the term ''galloping consumption'' suggests, acute pneumonic phthisis runs a short and stormy course, terminating fatally in children in the course of a few weeks, and in adult subjects in a few weeks or months.

In the absence of mixed infection, the disease is not attended by leucocytosis.

Physical Signs.—The physical manifestations of acute pneumonic phthisis, during the early stage of the disease, correspond in the main rather closely to those of lobar pneumonia, with certain differences in the distribution of the principal signs in the two diseases. In cases which survive for several weeks or for months, on the contrary, the clinical picture resembles that of the late stages of chronic ulcerative phthisis.

Inspection.—In acute pneumonic phthisis the respirations are rapid and shallow, and the patient sometimes exhibits such an extreme degree of asthenia as to be unable to inspire deeply upon request. Indeed, the inspiratory act is often so feeble that it is impossible to detect any difference in the expansion of the two sides of the thorax. Moreover, the disease may be attended by paroxysmal attacks of urgent dyspnea in which suffocation seems to be imminent. The cheeks commonly wear a hectic flush; and herpes labialis, which is so frequently observed in lobar pneumonia, is occasionally to be noted in acute pneumonic phthisis.

During the early days of the disease the sputum does not differ materially from that of atypical pneumonia. It is usually scanty, viscid, and occasionally has a rusty or bloody tinge. With the further progress of the disease the sputum becomes more abundant, less tenacious, and is apt to be streaked with blood. In certain cases frank hemoptysis occurs. Tubercle bacilli and elastic fibers are usually rather late in making their appearance, although in certain cases bacilli can be demonstrated during the first week of the disease.

Palpation.—Light finger-tip palpation of the upper thoracic region upon each side occasionally serves to reveal deficient expansion of the thorax upon the side of the disease. Palpation of the infraclavicular region during phonation is apt to elicit an exaggeration of vocal fremitus upon the side of the disease during the stage of consolidation. During the late stage, attended by extensive cavitation, the fremitus is exaggerated over the distribution of superficial cavities, which are surrounded by areas of consolidation.

The pulse is commonly accelerated to a degree which is markedly in excess of the pyrexia. The pulse is readily compressible; and, late in the disease, is frequently arrhythmic.

The systolic blood pressure is usually low in the presence of acute pneumonic phthisis, and this reading is attended, as a rule, by an abnormally low diastolic pressure.

Percussion.—During the early period of the disease, ascending percussion of the thorax usually reveals normal resonance or, indeed, a slightly hyperresonant note over the site of the disease, a finding which is succeeded by dullness due to consolidation as the disease progresses. In cases which survive to the stage of cavitation, percussion of the infraclavicular region occasionally yields a moderately tympanitic note; and it is sometimes possible to elicit a cracked-pot sound as well as one or more of the classical signs of pulmonary cavity.

Auscultation.—As a rule, the earliest auscultatory finding in this acute form of phthisis is a partial suppression of the vesicular murmur over one infraclavicular and upper interscapular region. With the progressive consolidation of the upper lobe, however, the respiratory sounds become bronchovesicular or frankly bronchial in character. This type of breathing persists for a week or ten days, when, instead of clearing as is the case in lobar pneumonia, signs of softening and excavation of the lung supervene. With the establishment of cavitation in the area of consolidation, the veiled puff can occasionally be detected, if a deep inspiration can be obtained.

Râles are not abundant in this form of phthisis, although fine crepitant râles are to be noted in cases undergoing rapid softening. Late in the course of the disease gurgling râles may occasionally be elicited over a pulmonary excavation upon inspiration or change of posture.

The heart sounds are commonly somewhat valvular, and the second sound at the pulmonary area is frequently accentuated. Reduplication of the second sound at the base is occasionally noted.

X-ray.—Radiographs may show areas of density resembling those seen in bronchopneumonia.

Diagnosis.—The physical signs of acute pneumonic phthisis rather closely simulate those of lobar pneumonia during the first two or three weeks of the disease. However, the consolidation of phthisis is commonly encountered in an upper lobe, whereas lobar pneumonia is prone to involve the base of the lung. Moreover, at the seventh or ninth day, instead of terminating by crisis with amelioration of the more acute symptoms, the disease pursues a hectic course, with night sweats, and the eventual appearance of elastic fibers and tubercle bacilli in the sputum. The appearance of hemoptysis or frank pulmonary hemorrhage speaks in favor of tuberculous disease, as does also absence of leucocytosis.

While the subject of acute pneumonic phthisis is often in apparently robust health at the time of the onset of the disease, careful questioning of the patient frequently will elicit the fact that for several weeks he has had a slight cough, with moderate loss of weight, some languor, and perhaps light nocturnal sweating. In any case in which signs of consolidation are detected in an upper lobe, a careful history should be taken with a view to bringing out these premonitory symptoms, which are immediately suggestive of phthisis.

It is to be borne in mind that a small percentage of cases of phthisis are complicated by a true lobar pneumonia. In this event the pneumonia habitually involves a lower lobe of the lung, and frequently is undetected during the life of the patient.

ACUTE BRONCHOPNEUMONIC PHTHISIS

Clinical Pathology.—Acute bronchopneumonic phthisis is the form of pulmonary tuberculosis which is most frequently encountered in infants and young children, although the disease attacks adults as well, especially female subjects following parturition. Cases have also been noted in adults afflicted with diabetes and in subjects of chronic alcoholism. Again, an acute tuberculous bronchopneumonia is occasionally the terminal event in the course of chronic ulcerative phthisis. In adult subjects of chronic phthisis a partially healed cavity may suddenly become active and result in a widespread acute bronchopneumonia of tuberculous origin.

The precise pathogenesis of the disease has not been conclusively established. The disease has been attributed to the rupture of a caseous lymph node in the hilus region, with discharge of its contents into a bronchus, as well as to the wide dissemination of the infectious contents of a rapidly formed tuberculous cavity in a subject with little resistance to the disease, a state which obtains in infants and young children. It has likewise been attributed to mixed infection with pyogenic organisms; but numerous cases have been examined in which no mixed infection could be demonstrated. The disease is frequently a sequel to an attack of pertussis or measles in a young child.

Acute bronchopneumonic phthisis is a bilateral disease, but the two lungs are seldom involved to an equal degree. As in other forms of phthisis, the lesions have their inception in the upper lobe in the vast majority of the cases.

The disease assumes the form of a caseous bronchopneumonia, the areas of consolidation occupying a radial distribution around the involved bronchioles. The patches may be quite small and separated by bands of crepitant pulmonary tissue, or large lesions may occur as a result of the fusion of adjacent areas. When such a conglomerate area of consolidation forms rapidly, caseation frequently occurs in the midst of the lesion, with consequent cavity formation. Very acute cases not infrequently terminate fatally before excavation of the lung supervenes, whereas in the cases which survive for a few weeks cavitation is the rule. The disease extends rapidly, involving a large portion of a lobe, and occasionally an entire lung.

As a general rule, there is no effort to wall off the area of consolidation by fibrous tissue, so that the diseased area shades off almost imperceptibly into the adjacent pulmonary tissues. In rare instances, however, moderate fibrosis occurs, and in these cases the further course of the disease is rather similar to that of chronic ulcerative phthisis.

The pleura investing the portion of the lung which is involved in the disease is commonly covered with an acute plastic exudate; but effusion into the pleural cavity is not a frequent finding at autopsy.

In the majority of the cases of tuberculous bronchopneumonia the onset of the disease is insidious, with cough, moderate elevation of temperature, anorexia and languor. In other instances the onset is abrupt, occasionally with an initial hemoptysis. The disease usually runs a short course of a few weeks, during which the temperature remains elevated, and emaciation becomes extreme. In rare instances, instead of terminating fatally in a few weeks, the disease assumes a fibroid form, and the further course resembles that of chronic phthisis.

Physical Signs.—*Inspection.*—The subject of bronchopneumonic phthisis presents a degree of dyspnea which usually exceeds that attending nontuberculous bronchopneumonia. The expansion of the thorax is trivial, and transient suffocative attacks are not infrequent. Such paroxysms are attended by considerable degrees of cyanosis of the mucous membranes and digits. With the further progress of the disease cough becomes distressing, the cheeks wear a hectic flush, and emaciation becomes a striking feature of the case. In late stages of the disease the patient is usually dull and somnolent, with dry lips and tongue, and a general appearance of mental torpor. In other cases terminal convulsions occur.

Palpation.—Light finger-tip palpation of the upper thoracic region may reveal a slight lagging of one apex during an early stage of the disease. With the full establishment of the bronchopneumonia, however, thoracic expansion is so slight that it is difficult to detect any unilateral discrepancy in expansion. Palpation of the infraclavicular and mammary regions frequently serves to reveal a moderate exaggeration of vocal fremitus.

The pulse in acute bronchopneumonic phthisis is accelerated beyond the degree of pyrexia, and is commonly small and readily compressible. Various disturbances in rhythm may be encountered during the late stage of the disease.

As in other forms of phthisis, a low systolic blood pressure usually obtains in tuberculous bronchopneumonia, and it is commonly attended by a commensurate diminution in the diastolic pressure.

Percussion.—Ascending percussion of the thorax during the early days of the illness usually reveals a mildly hyperresonant note in the upper mammary, infraclavicular, and upper interscapular regions. With the further evolution of the disease this note is succeeded by a variable degree of impairment of resonance in these regions, which seldom attains to distinct dullness. Similarly, ascending percussion of the lateral thoracic wall, usually elicits similar signs in the upper axillary region. During the late stages of cases which are attended by extensive excavation of the lung, percussion of the infraclavicular region may reveal tympany of moderate degree, or a cracked-pot sound upon forcible percussion.

Auscultation.—Auscultation of the superior thoracic regions is apt to reveal a prolongation of expiration or frank bronchovesicular respiration during the stage of consolidation of bronchopneumonic phthisis. It is rare that the consolidation is so dense as to be attended by distinct bronchial respiration. Subcrepitant râles are frequently to be elicited in the same regions; and, with the full development of the bronchopneumonia, crepitant râles upon the completion of inspiration become audible. In late cases, which are attended by extensive cavitation, gurgling râles are occasionally to be elicited, and the respiratory sounds are apt to assume a cavernous or amphoric character.

The cardiac sounds are commonly valvular in quality, with accentuation or reduplication of the second sound at the pulmonary area. During late stages of the disease embryocardia may be present, or various disturbances of cardiac rhythm may be in evidence.

X-ray.—The x-ray findings are usually those of a chronic pulmonary tuberculosis, with infiltration of one or both apices and considerable pleural thickening. In addition, there may be several small areas of increased density giving the lung a mottled appearance. These spots range in size from one to five millimeters in diameter.

Diagnosis.—The extreme degree of prostration of the patient and the rapid march of the disease readily place acute tuberculous bronchopneumonia above the gravity of an acute bronchitis.

The principal problem in the majority of cases is to differentiate the disease from a nontuberculous bronchopneumonia. Any bronchopneumonia which has its inception in the upper lobe, and in which the principal physical signs are elicited in the upper axillary, mammary and infraclavicular regions, is strongly suggestive of phthisis. The rapid emaciation of the patient points to the same origin of the disease, while the development of a persistent tachycardia which is in excess of the degree of pyrexia is suggestive of phthisis. Hemoptysis is frequently an early symptom, and tubercle bacilli can be detected in the sputum during the early days in many cases. X-ray of the thorax frequently is of distinct aid in diagnosis; and the tuberculin test may prove of value in a young subject.

ACUTE MILIARY TUBERCULOSIS

Clinical Pathology.—This form of acute phthisis is encountered most frequently in young subjects, but no age is exempt from the disease. It is initiated by the widespread dissemination of tubercle bacilli by the blood stream, with the consequent induction of miliary tubercles in widely separated organs of the body.

The bacillus may gain entrance to the blood stream through the rupture of a caseous tuberculous gland, from a focus in the lower intestinal tract through the thoracic duct, or through the formation of tubercles in the walls of blood vessels with subsequent discharge of bacilli through the endothelial lining of the vessel. In either event a tuberculous bacteremia results, which in itself seems not to be an adequate cause of acute miliary tuberculosis, as a tuberculous bacteremia occasionally is noted without the development of miliary infection. It would seem that in the induction of the disease there must be a coincident diminution of certain undetermined protective properties of the blood serum. This hypothesis is borne out by the frequency with which miliary tuberculosis follows childbirth, surgical operations, acute infections such as measles and pertussis, and exposure to inclement weather, in subjects of arrested or localized chronic phthisis.

The lesions of acute miliary tuberculosis occur in the lungs, which the disease seems to involve by predilection, and also in more remote organs, notably the liver, spleen, kidneys, bone-marrow, meninges, thyroid gland, and the choroid coat of the eye. The principal distribution of the lesions sometimes affords some light as to the probable site of the original focus of infection. Thus, in cases which are largely limited to the lung, without involvement of distant organs of the body, the causative focus is usually a caseous bronchial gland or a partially healed tuberculous cavity. When, on the other hand, the principal disease is limited to the liver, the initial focus is usually situated in the intestinal tract, which is drained by the portal vein. Similarly, a uniform distribution of the disease throughout both lungs points to the areas drained by the radicles of the thoracic duct as the probable source of infection.

Three forms of acute miliary tuberculosis are recognized; namely, the *pulmonary* form, with which this work is directly concerned, the *meningeal* form, and the *typhoid* form, of the disease.

In the *pulmonary* form of acute miliary tuberculosis the lungs are dark red in color, and their total bulk is increased. Upon section, the surface of the section is dark red, and is studded with small macroscopic conglomerate tubercles. The older lesions appear upon the hyperemic surface of the lung as small grayish-white or yellow nodules, while more recent tubercles occur as minute, grayish, semitransparent lesions. In cases which complicate a former tuberculous focus in the lung, the tubercles are most numerous in the region immediately contiguous to the causative focus, and gradually shade off toward the periphery of the lung.

Excavation of the lung is most exceptional in this acute form of phthisis, on account of the rapid downward course of the disease.

The bronchial glands are enlarged, sometimes calcified from a former infection, and not infrequently are caseous. The bronchial mucous membrane presents an active hyperemia, and varying grades of acute catarrhal inflammation are in evidence.

The pleura investing the diseased lung is frequently studded with small macroscopic tubercles, and may be the seat of a plastic exudate. Pleural effusion is an occasional accompaniment of the disease. In cases which survive for several weeks tubercles are demonstrable in the meninges, peritoneum, and occasionally in the choroid coat of the eye.

The onset of acute miliary tuberculosis is abrupt, with a rise of fever, cough, and pain in the side, representing a sudden turn for the worse in a subject of chronic phthisis, or developing in a child after an attack of pertussis or measles. In the pulmonary form of the disease expectoration is present, assuming a mucopurulent character, sometimes containing blood and tubercle bacilli. The fever usually remains relatively high throughout the course of the disease, although remissions and exacerbations sometimes are noted. The disease commonly terminates fatally in eight to twelve weeks. During this period a transition from one form of the disease to another may sometimes be observed. It is not uncommon to see a case which began as a purely pulmonary disease assume the typhoid or the meningeal form later in its course.

Physical Signs.—*Inspection.*—The physical manifestations of acute miliary tuberculosis of the lungs are somewhat similar to those of acute catarrhal bronchitis, with however, a more extreme degree of prostration. Dyspnea is a striking feature of the case, and cyanosis frequently develops upon the slightest effort or even with the patient in repose. The respirations are shallow and occasionally attain a frequency of 70 or 80 per minute in young subjects. There is no visible unilateral variation in expansion of the thorax, but rather a bilateral restriction of this function. Litten's sign is usually diminished in extent or absent.

Cough is persistent, and is attended by the raising of mucopurulent sputum, which early in the course of the disease may become sanguineous. Tubercle bacilli may or may not be demonstrable in the sputum.

Palpation.—Palpation of the thorax in acute miliary tuberculosis seldom yields any definite diagnostic data. Pleural friction fremitus may be elicited in a limited number of cases, usually over the superior regions of the chest.

The pulse is unduly accelerated from the onset of the disease, and it continues to augment in rate until the fatal issue. Counts of 160 or 180 beats per minute are not uncommon.

Palpation of the upper abdominal quadrants very frequently reveals enlargement of the spleen or of the liver.

Percussion.—In acute miliary tuberculosis of the lungs the findings upon percussion of the thorax are variable. As a rule, the note is moderately hyperresonant, despite the wide dissemination of miliary tubercles throughout the lungs. In the case of children, in whom the disease has arisen as a sequel of measles or pertussis, the percussion findings are very similar to those of nontuberculous bronchopneumonia, with scattered areas of dullness over the bases and Skodaic resonance in the infraclavicular and mammary regions. In cases which are attended by pleural effusion dullness or flatness is elicited over the dependent portion of a lung.

Auscultation.—Moist râles are demonstrable over both lungs, becoming more numerous and assuming the mucous type toward the termination of the disease. Pleural friction is occasionally encountered; and Jürgensen has described a fine crepitation which is caused by the presence of miliary tubercles upon the pleura. In children the breath sounds over the bases posteriorly are occasionally bronchovesicular in type.

X-ray in the early stages may be negative, later the whole pulmonary surface may be covered with fine specks as though salt or pepper had been sifted over the lung. Evidence of consolidation may be present from an old lesion. The bronchial gland shadows are usually increased.

Diagnosis.—The urgent dyspnea and the degree of prostration of the patient serve to place the gravity of the disease above that of a simple catarrhal bronchitis of nontuberculous origin. In the case of children it is often difficult for a time to differentiate the disease from a nontuberculous bronchopneumonia. The eventual appearance of tubercle bacilli in the sputum, and positive tuberculin tests serve to establish the tuberculous origin of the disease.

In the adult subject the disease is readily confused with typhoid fever, as in the typhoid form of acute miliary tuberculosis the two diseases bear numerous points of similarity. As a rule, however, these subjects are known to be afflicted with chronic phthisis, which should arouse a suspicion of a possible terminal miliary infection with the tubercle bacillus. Moreover, the specific tests for typhoid fever prove negative. The combination of leucopenia with a moderate increase in polymorphonuclear neutrophiles should suggest miliary tuberculosis rather than nontuberculous septicemia.

In the meningeal form of acute miliary tuberculosis headache is one of the cardinal symptoms of the disease. As tuberculous patients, even in the presence of fever, seldom complain of headache, this symptom alone is suggestive of meningeal involvement. Likewise, retention of urine in a patient showing a tendency toward somnolence during a late stage of phthisis is an important sign.

If the eyes are carefully examined in a patient suffering with the meningeal form of acute miliary tuberculosis, inequality of the pupils, a sluggish reaction to light, strabismus, nystagmus, or double vision may be noted.

Spinal puncture commonly reveals a clear fluid, which is under moderate pressure, and which usually shows a moderately increased cell count. Tubercle bacilli are rarely demonstrable in the fluid, but guinea pig inoculation is sometimes positive in otherwise negative cases.

CHRONIC ULCERATIVE PHTHISIS

Clinical Pathology.—Chronic ulcerative phthisis is the most frequent form of pulmonary tuberculosis which is seen by the clinician. This form of tuberculosis attacks females with slightly greater frequency than male subjects, usually manifesting its presence during the third or fourth decade of life, although cases are noted at earlier and later age limits. The primary involvement is most frequently situated in an upper lobe, although in children the initial lesion may be located in the hilus region or even in the lower lobe.

The predominant tissue changes in chronic ulcerative phthisis are of the productive or proliferative type, rather than of the exudative type which is observed in acute pneumonic phthisis, or acute caseous pneumonia. The miliary tubercle, the primary lesion of the disease, is far too minute to produce or to modify the physical signs of the disease. Of greater interest to the clinician is the conglomerate tubercle, a larger lesion, formed by the coalescence and fusion of a number of adjacent miliary tubercles.

The site of this lesion is peribronchial, and it originally has no communication with the bronchial system, hence it follows that any physical manifestations of the presence of such a lesion upon auscultation must depend not upon the presence of exudation or moisture in the bronchioles or alveoli, but upon compression of these structures. Hence, the auscultatory findings at this early stage of the disease are apt to consist largely of dry sibilation. However, it is conceivable that extensive peribronchial lesions may be attended by mild bronchial irritation, producing moist sounds upon auscultation, or indeed, even exciting cough and expectoration. In such cases tubercle bacilli cannot be demonstrated in the sputum. This closed case becomes an open one, with tubercle bacilli in the sputum, when the peribronchial infiltration caseates and the bronchiolar wall is eroded.

The predilection of the tubercle bacillus for the apical portions of the lungs has not been adequately accounted for, any more than has the predilection of the organism for pulmonary tissue. It is assumed to be due to the fact that the apices of the lungs are less freely mobile than are the lower lobes, resulting in a relative retardation of the flow of the inspired air, as well as of the blood and lymph streams in these regions, a state of relative repose which favors infection with the tubercle bacillus. Cobb attributes the greater frequency of apical infection to the defective glandular supply of this portion of the lung, while Freund attributed it to stenosis of the superior aperture of the thoracic cavity by a pathologic shortness of the first rib.

Fowler, in an extensive autopsy experience, found the initial focus of infection localized most frequently at a point from one to one and a half inches below the extreme pulmonary apex, and nearer the external and posterior borders of the lung, whence the disease progressed backward toward the suprascapular region. Anteriorly, the primary lesion was found to correspond to the inner third of the supraclavicular fossa. With less frequency the initial focus was encountered at a greater distance from the apex, occupying a point corresponding to the first and second intercostal spaces, below the outer third of the clavicle. The direction of extension of the disease in these cases was downward. The third point of predilection for the primary lesion was found to be the apex of the lower lobe, at the level of a point upon the chest wall opposite the fifth dorsal vertebra, midway between the vertebral column and the scapula. From this point of inception, the disease progressed posteriorly as well as laterally.

The opposite lung was seldom found involved in the absence of disease of the lower lobe of the lung which was originally infected. When the opposite lung was involved, the distribution of the lesions was similar and their progression was along the same lines as in the lung primarily infected. In its progression the tuberculous infiltration proceeds in an irregular manner, leaving areas or bands of crepitant pulmonary tissue in the area of involvement, which modify and, to a certain degree, tend to mask physical signs caused by the tuberculous infiltration.

As a natural consequence of the histologic structure of the tuberculous infiltration, as the mass extends by peripheral growth the central portion, representing the original conglomerate tubercle, becomes ischemic and tends to undergo softening and caseation. The usual termination of an area of caseation is

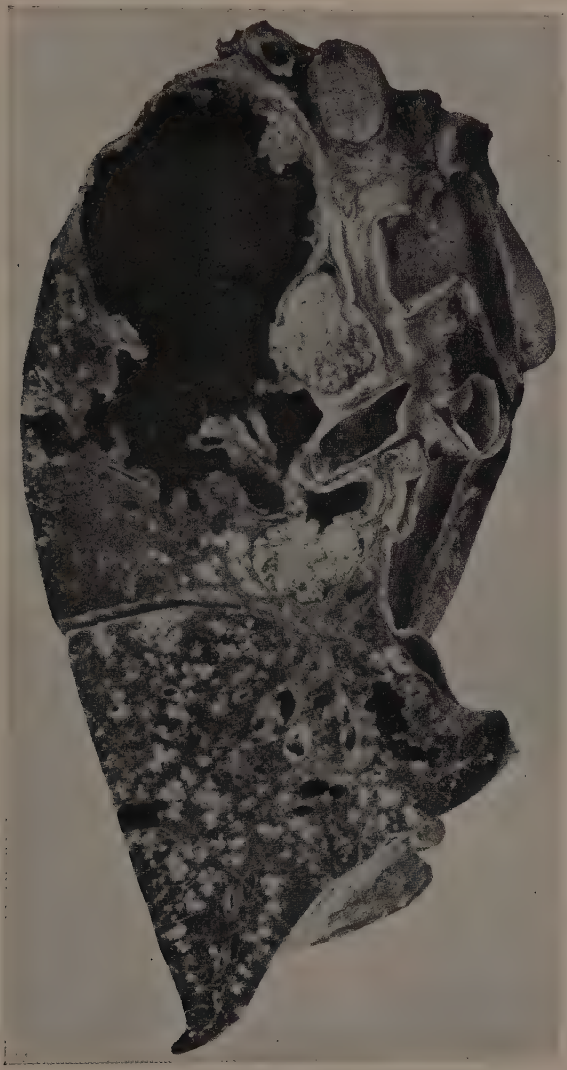

Fig. 284.—Lung. Chronic phthisis, showing a large irregular cavity in the upper lobe. In the lower lobe there are scattered acute nodules grouped in clusters around the small bronchi; and also several small more acute cavities. The bronchial glands are enlarged and caseous. (Edingurgh University Anatomical Museum.) (Woolley after Beattie and Dickson.)

pulmonary excavation with the production of a cavity, containing caseous material which, by admixture with serum, constitutes the so-called tuberculous pus. That these destructive lesions are formed quite rapidly is evinced by the fact that with proper staining methods the remnants of the original elastic tissue structure of the lung can be demonstrated in these areas of caseation. Under favorable conditions, areas of caseation may be replaced by fibrous tissue, constituting healing by sclerosis.

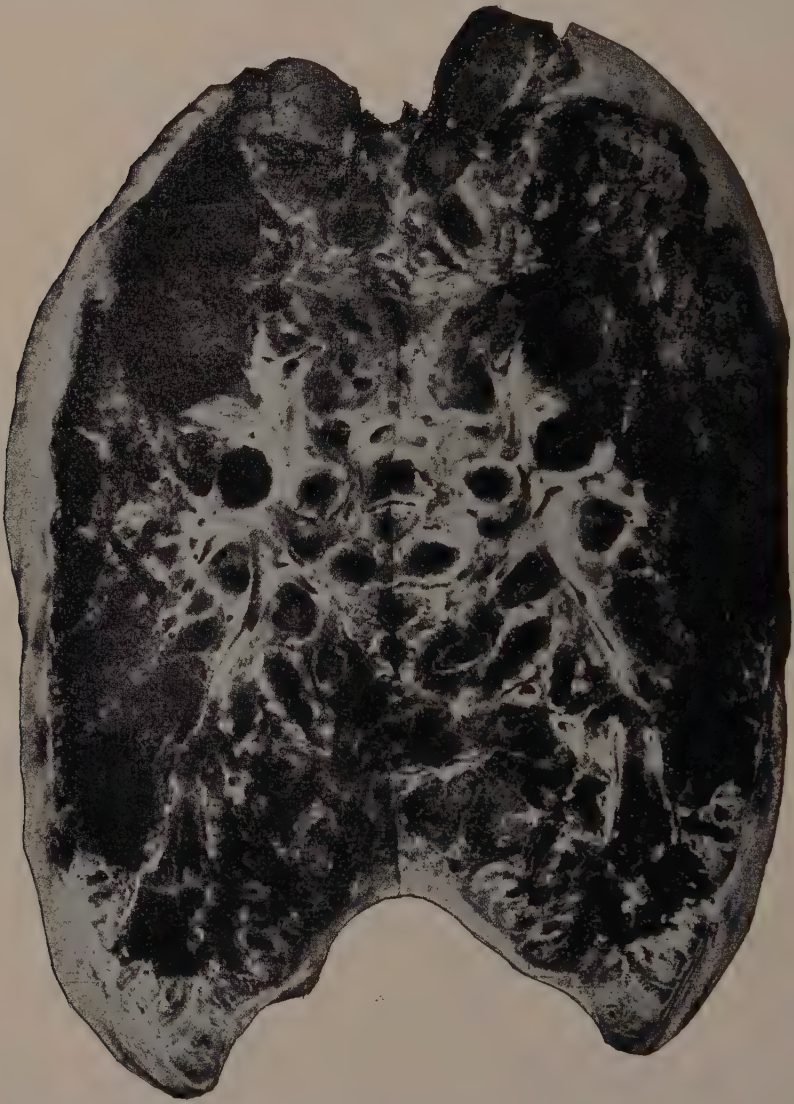

Fig. 285.—Illustrating pulmonary tuberculosis, with thickened pleura, many bronchiectatic cavities, and generalized cavity formation. (Pottenger, after Tendeloo.)

It is well established that whereas in the case of solid organs infection with the tubercle bacillus is usually followed by the formation of a solid tuberculoma, in the case of the lungs the usual tendency is toward destruction of tissue and cavity formation. These cavities may be single or multiple, and of different shapes and dimensions. When multiple cavities form, the intervening walls are occasionally destroyed, forming large excavations which may embrace a large part of a lobe or, indeed, in rare instances an entire lung.

The distribution of the cavities in chronic phthisis is not constant. The greater number occupy the upper lobe toward the pulmonary apex; but Ewart has shown that cavitation usually appears in the apex of the lower lobe before excavation involves the inferior portion of the upper lobe. In the case of the right lung, moreover, in the downward progression of the disease, the middle lobe is most frequently passed over, the morbid process extending directly from the upper lobe to the apex of the lower lobe of the involved lung. The bases of the lungs and the anterior borders of the lower lobes are seldom the seat of tuberculous cavities.

There has been much discussion as to the probable rôle of mixed infection in the formation of cavities in this disease. While this factor is probably operative in certain cases of pulmonary excavation, experimental and clinical work has shown that the tubercle bacillus alone can and does cause extensive cavitation in the absence of pyogenic infection.

The frequency with which bronchiectatic cavities occur in chronic phthisis is likewise an unsettled question. While bronchiectatic cavities occur in this form of the disease, they are comparatively infrequent as compared to their frequency in the fibroid form of chronic phthisis. In ulcerative phthisis the vast majority of cavities arise as a result of pulmonary infiltration with subsequent caseation and expulsion of the necrotic tissues by expectoration.

The natural tendency of the phthisical cavity is to extend slowly by peripheral invasion; but occasionally the proliferation of connective tissue around the cavity serves to limit its spread and actually may cause the cavity to shrink. This reparative sclerosis tends to limit the peripheral growth of the cavity; but, when there is a patent communicating bronchus, the disease may continue to spread as a result of bronchogenic metastasis. In certain instances the bronchial communication is permanently obliterated, and a truly closed cavity is formed. More frequently, however, the bronchus is merely plugged with necrotic material, producing a potentially closed cavity.

Large pulmonary cavities are not infrequently traversed by trabeculæ of partially necrotic pulmonary tissue or by branches of the pulmonary artery. As a rule, the lumen of such a vessel is obliterated; but in certain instances the lumen remains patent; and, upon erosion of the vessel wall, copious or fatal hemorrhage may ensue.

When a peripheral portion of the lung is the seat of a rapidly extending cavity, the investing pleura may be perforated, with the induction of spontaneous pneumothorax. The subsequent entrance into the pleural cavity of infectious material from the pulmonary cavity usually results in the development of a fatal pyopneumothorax. In the case of slowly developing peripheral cavities, on the contrary, adhesions may be established between the visceral and the parietal pleura, which serve to prevent this serious complication.

The pleura is involved in nearly every case of chronic phthisis. The membrane may be merely thickened; there may be fine adhesions which are readily torn asunder; or dense bands of adhesions may unite the visceral to the parietal pleura. Pleural changes are most frequently encountered over an upper lobe, but they are also seen in the interlobar pleura, as well as over the diaphragm, in the latter event serving to limit the mobility of the lung in this direction. Tuberculous pleurisy may assume the acute fibrinous form; but pleural effusion is not infrequent; and, in cases of extensive duration, the findings may approach those of chronic adhesive pleurisy.

As the tuberculous infiltration progresses, the uninvolved portions of the lungs become the seat of compensatory emphysema. In the presence of extensive destruction of one lung, the anterior border of the opposite lung frequently passes the median line, while the voluminous base depresses the diaphragm, which is compensatorily elevated upon the side of the diseased lung. In the case of bilateral apical infiltration, the anterior borders and the bases of the lungs exhibit a condition of compensatory emphysema.

Similarly, in the case of extensive unilateral disease, with diminution of the total bulk of the lung, the mediastinum is displaced toward the affected organ by the pressure of a compensatory emphysema of the opposite lung. Under these circumstances the heart is displaced toward the side of the disease, and the trachea and larynx are similarly deflected.

Aside from the mechanical displacement of the heart which occurs in unilateral disease, this organ is usually atrophic in chronic ulcerative phthisis, and it tends to occupy a vertical position when mediastinal shifting does not occur. It is frequently the seat of chronic myocarditis as a result of toxic absorption. Pericarditis with effusion is occasionally noted, as is also a verrucose type of endocarditis. Miliary tubercles of the myocardium, which occur in certain cases of acute miliary tuberculosis, are practically never seen in chronic phthisis.

The larynx becomes involved in approximately one-third of all cases of chronic ulcerative phthisis, the extension of the disease occurring by way of the lymphatics rather than as a result of local inoculation by bacilli borne in the sputum. The initial change is the formation of miliary tubercles subjacent to the mucous membrane. Through softening and caseation, irregular ulcers are ultimately formed upon the mucous surface. In these changes the epiglottis, aryepiglottic folds and vocal cords are the points of predilection of the lesions.

The bronchial glands are early involved in the tuberculous infection. Infiltration, calcification, and abscess formation with rupture of the glands have all been noted. A caseous gland in close proximity to a large vein is a potent source of danger, as it may adhere to the vessel, and, by subsequent rupture, may discharge its contents into the circulation with the induction of acute miliary tuberculosis.

Further possible complications of chronic ulcerative phthisis include tuberculosis of the genitourinary organs, female reproductive organs, gastrointestinal tract, bones and joints, peritoneum, nervous system, and amyloid degeneration of various organs, notably of the liver, kidneys, spleen, and intestine.

Chronic ulcerative phthisis in its evolution and course produces no changes in the cytology of the blood which are pathognomonic of the disease. In the majority of cases the hemoglobin percentage remains practically normal until a late stage of the disease, when a secondary hemoglobinemia frequently becomes manifest. The total number of erythrocytes is little altered during the early period of the disease. Indeed, some clinicians have noted a polycythemia at this time. Advanced cases, however, associated with mixed infection, frequently exhibit a diminution of red cells coincidently with a like falling off in the hemoglobin. As a general rule, in early phthisis the leucocyte count shows no deviation from the normal, although isolated cases show a moderate leucocytosis at this time. In advanced cases, on the contrary, associated with cavitation and mixed infection, moderate leucocytosis is frequently noted.

In the early stages of chronic ulcerative phthisis the urine contains no elements which are characteristic of the disease. During late periods of the disease albuminuria and occasional casts are encountered, and seem to be due to irritation of the kidneys by tuberculous toxins rather than to an acute nephritis.

The onset of chronic ulcerative phthisis is insidious, with progressive loss of weight, ready fatigue upon moderate exertion, dyspeptic symptoms, menstrual disturbances, light nocturnal sweating, and a general feeling of languor. In certain cases the first intimation of the disease is a pleural pain localized over the superior thoracic regions, or a persistent hoarseness. In other cases the disease is ushered in with an initial hemoptysis.

As the name implies, this form of phthisis follows a rather prolonged and indefinite course, usually with remissions and exacerbations of the clinical symptoms of the disease. In the case of patients in whom the hygienic surroundings are not proper, and in whom proper feeding and rest cannot be obtained, the span of life is often little more than a year from the onset of the disease. In cases which undergo the proper régime, however, the disease pursues a lengthy course, and permanent arrestment of the disease is frequently obtained.

The ultimate prognosis of the individual case depends to a great degree upon the integrity of the heart muscle. Unfavorable symptoms embrace high temperature, pulse rates which are out of proportion to the degree of pyrexia, loss of weight which cannot be replaced by abundant feeding, and involvement of the larynx or gastrointestinal tract in the disease.

Physical Signs.—The physical manifestations of chronic ulcerative phthisis present a wide range of variety, depending as they do upon the duration and progress of the individual case, and upon the physical makeup of the patient prior to his infection. All gradations are encountered, from the truly early case with normal thoracic contour, integument and subcutaneous tissues, presenting no definite physical signs of the disease, to the advanced case which presents signs which the layman recognizes as characterizing tuberculosis of the lungs.

Inspection and Palpation.—During the examination of a case of suspected phthisis, the examiner should not limit his inspection to the thorax; but a careful inspection of the entire body should be practiced, taking into account the face, neck, abdomen, and extremities. Clinicians of the present day do not attach much significance to the presence of the paralytic or alar thorax in chronic phthisis. While this thorax is encountered frequently in this disease, the other classical types are so often seen that the so-called phthisical thorax has lost much of its diagnostic importance. Many subjects of chronic phthisis present a partially developed emphysematous thorax, and others frequently present the rachitic thorax, the funnel-chest, or Harrison's sulcus, without any apparent causal connection with their disease. Rounding of the shoulders and spinal curvature are of greater diagnostic importance than is a long and flat thorax. This rather characteristic rounding of the shoulders, frequently associated with depression of the infraclavicular regions, represents an attempt upon the part of nature to splint the diseased upper lobe.

In many early cases inspection of the supraclavicular regions proves negative. In other instances, however, the fossæ may be of unequal depth, a sign which does not invariably signify disease of the underlying apex. Light palpation of these regions with the finger-tips may show, in the presence of a unilateral apical lesion, lagging of the corresponding supraclavicular region. Under these circumstances it is observed that the expansion of the affected side is delayed, or that it does not attain the degree of expansion of its fellow upon the opposite side. In the presence of bilateral lesions of the upper lobes, this mode of examination frequently proves fallacious, yielding findings which require percussion and auscultation of the areas in order to secure conclusive data.

Undue prominence of the clavicle upon one side, when considered in conjunction with muscle spasm or degeneration, may betray an active or latent apical lesion. Frequently there is moderate dropping of the shoulder upon the side of the disease, whereupon a line drawn from the base of the neck to the tip of the shoulder is longer than is a similar line erected upon the opposite side; and, moreover, the convexity of the line upon the side of the disease is directed outward.

Inspection of the anterior surface of the thorax in an early unilateral case sometimes reveals deficient expansion of the affected side, most noticeable above the level of the fourth rib. Frequently the deficiency is so slight that it is best elicited by drawing a line upon the two sides immediately below and parallel to the clavicle, and inspecting the thorax over the shoulders of the patient. Under these conditions during inspiration the line comes into view sooner upon the unimpaired side of the thorax. In the truly incipient case of chronic phthisis this deficiency of expansion is to be attributed to muscle spasm rather than to destruction of pulmonary tissue or to pleural adhesions.

In the advanced case, on the contrary, an entire side may exhibit little expansion, in striking contrast to the vicarious expansion of the opposite compensatorily emphysematous side. In this event the examiner should search for signs of displacement of the mediastinum. In the case of an extensive destruction of the right lung it is not uncommon to find the cardiac impulse displaced to the right of the sternum. In this connection it is to be remembered that pulmonary excavation and collapse are attended by displacement of the mediastinal contents toward the side of the lesion, whereas pleural effusion and pneumothorax cause displacement of these structures in a direction away from the lesion.

Upon the lateral aspect of the thorax, in the presence of extensive unilateral disease, the ribs are unduly prominent, as a result of atrophy of the intercostal muscles and subcutaneous tissues. The intercostal spaces are narrow, and the costal angle is unduly acute.

Litten's sign is little affected in the early stages of the disease, but with extreme consolidation it is usually shortened and may be entirely absent on the side most affected.

Upon the posterior thoracic surface, in unilateral phthisis, there is noted spasm or atrophy of the suprascapular muscles upon the side of the disease, causing an apparent prominence or flattening of this region; and the scapula commonly occupies a position nearer to the vertebral column than is the case in the normal subject. At the same time the inner border of the bone frequently stands out prominently, and the scapula is readily displaced from its bed. In the presence of bilateral apical disease there is apparent prominence or flattening of both suprascapular regions, arising as a result of reflex spasm or atrophy of the subjacent muscles. In such a case the findings upon inspection and palpation must be abetted by those obtained upon percussion and auscultation of the thorax.

In the presence of a dense apical consolidation, vocal fremitus is apt to be increased in intensity; but very many of these cases are attended by pleural thickening which effectually obscures any exaggeration of fremitus. Also, occasionally a large pulmonary cavity with a patent bronchial communication may be situated in the periphery of the lung, and afford an exaggeration of vocal fremitus over its distribution. Over the distribution of pleural effusions, extensive pneumothorax, or excessive pleural thickening, on the contrary, the vocal vibrations are diminished in intensity or are abolished.

The facies of early phthisis frequently presents no deviation from the normal, save for an unusual brilliance of the eyes. With the further progress of the disease, however, the facial aspect is apt to become markedly altered, partially as a result of muscular atrophy, and partially through reflex phenomena. In this class of case the malar bones are unduly prominent; the cheeks are more or less sunken, occasionally with a hectic flush corresponding to the side of the major lesion; the lips are usually thin, frequently retracted, and sometimes cyanotic; while, in contrast with the small face, the ears seem to be abnormally large for the individual.

In such a facies, as a result of wasting of the post-orbital adipose tissue, the globes tend to recede unduly into the orbits. The palpebral fissure upon the side of the disease also appears unduly wide, as a result of atrophy of the fibers of the orbicularis palpebrarum muscle. The diameter of the pupils should be carefully measured, as the pupil upon the side of the disease in unilateral cases is occasionally dilated to a variable degree.

Upon palpation of the structures in the median line of the neck, lateral deviation of the trachea may be made out in many cases of unilateral disease. The position of the trachea is readily determined by placing the palms upon the shoulders at the base of the neck, and gently insinuating a thumb between the trachea and the sternomastoid muscle upon either side.

Moderate enlargement of the thyroid gland is frequently noted in subjects of chronic phthisis.

The cervical muscles, especially the sternomastoid, scalenus anticus, and trapezius, should be inspected and lightly palpated for spasm or degeneration. Pottenger has shown that over an old quiescent apical lesion atrophy of these muscles together with the skin and subcutaneous tissues will be revealed by a lack of elasticity upon light palpation, and that the separate muscular bundles may frequently be separated by the palpating fingers. In the presence of an active lesion, on the contrary, the same muscles impart a sensation of increased tension to the palpating fingers, and may stand out more prominently than their fellows upon the opposite side.

When the larynx is involved in the tuberculous process, the pharyngeal mucosa is commonly pallid, although there are exceptions to this general rule. Uniform redness of both vocal cords may be encountered, although redness of a single cord is more pathognomonic of tuberculous laryngitis. The epiglottis may be enlarged to such a degree as to obscure the view of the interior of the larynx. Mere redness of the laryngeal structures, however, does not constitute a diagnostic sign of tuberculous laryngitis, as this change may be induced by reflex disturbances.

Clubbing of the fingers and incurvation of the nails are to be noted in a good many cases of chronic phthisis, although these findings are more common in bronchiectasis. The nails may present longitudinal or lateral incurvation; and, in very far advanced cases, the hands and feet may present the deformities recognized as hypertrophic pulmonary osteoarthropathy. In late cases edema of the extremities may be encountered as a result of gradually developing cardiac insufficiency, while certain cases present a characteristic sweating of the ankles a few weeks before a fatal issue of the disease.

The pulse in chronic ulcerative phthisis presents numerous variations. In certain early cases it is but moderately increased in frequency, and it is sometimes even slowed. In febrile cases, as a rule, however, the acceleration of the pulse is out of proportion to the degree of pyrexia. Many cases present a continuous tachycardia, while others are subject to paroxysms of rapid heart action, which are indistinguishable from paroxysmal tachycardia in a nontuberculous individual.

In the presence of complicating peritoneal or intestinal tuberculosis, the abdominal contour may be altered as a result of effusion into the peritoneal cavity. In certain late cases a similar enlargement is caused by general anasarca. In yet other instances the termination of the disease is preceded by abdominal distention due to extreme tympanites, which is obstinate and refuses to yield to measures adopted to reduce it.

In the earliest stages of the disease, chronic ulcerative phthisis is attended by little or no sputum. The earliest sputum consists largely of viscid mucus, which may be attended by froth. The expectoration floats when placed in water. With the further advance of the disease, yellowish streaks are to be encountered in the sputum, which later becomes mucopurulent and is frequently streaked with blood. During the advanced stage of the disease the sputum is commonly thin, dark gray or greenish in color, and contains numerous tubercle bacilli, elastic fibers, and various associated bacteria. It is uncommon to encounter undue fetor in the sputum of chronic phthisis.

Percussion.—In the early case of chronic ulcerative phthisis percussion of the thorax is apt to reveal alterations in the note elicited over the supraclavicular, infraclavicular, suprascapular, and upper interscapular regions. In this disease it is sometimes advisable for the examiner to begin his percussion of the thorax inferiorly and to work upward toward the apex, employing at first relatively forcible percussion and substituting light superficial stokes as the apices are approached. In this manner he works from a normal percussion note toward an abnormal sound, which may be moderately hyperresonant or dull, according to the duration of the disease.

It is a grave mistake to anticipate a constant dulling of the percussion note over the upper lobe in dealing with cases of incipient phthisis. Frequently at this stage of the disease the dissemination of the areas of infiltration and the vicarious distention of the intervening areas result in a mildly hyperresonant note over the superior regions of the thorax. In the more advanced case, however, percussion over the upper lobe frequently reveals an area of defective resonance. Such areas are usually encountered in the infraclavicular region below the inner third of the clavicle, or in the interscapular region above the scapular spine. In percussing the anterior thorax, the examiner should bear in mind that vesicular resonance is slightly impaired in the right infraclaviular region in the normal subject, and that an equalization of the note upon the two sides may possess potent diagnostic significance.

In every case of suspected phthisis the examiner should delimit and mark upon the chest wall the limits of pulmonary resonance at the apices and the bases. As a general rule, early in the course of the disease this method of examination will reveal a limitation of resonance over the apex of the affected side. Under these circumstances Krönig's isthmus upon the corresponding side is usually less than 2 inches in width; and percussion of the base of the lung will usually reveal a limitation of its descent during inspiration.

In the advanced case of chronic phthisis more extensive dulling of the percussion note is encountered in diverse regions of the thorax; and the interpretation of the findings is attended by greater difficulty than in the incipient case, as these findings may depend upon apical consolidation, thickened pleura, pulmonary collapse, pleural effusion, or pyopneumothorax. At this stage of the disease dullness of one or both sides to the fourth rib anteriorly and to the scapular spine posteriorly commonly points to extensive consolidation of the upper lobes. Krönig's isthmus may be totally obliterated upon the side of more extensive involvement and markedly narrowed upon the opposite side. Flatness in Traube's semilunar space in these late cases indicates pleural effusion or pyopneumothorax, while an extension of gastric tympany in the same area points to retraction of the anterior border of the left lung or to pleural adhesions.

These signs merely indicate the presence of retrogressive changes in the bronchopulmonary system. They afford no conclusive means of differentiation between active phthisis, healed tuberculous lesions, chronic interstitial pneumonia, pleural thickening, and fluid in the pleural cavity. In these advanced cases a careful study of muscle spasm and degeneration and fluoroscopy of the thorax frequently serve to reveal the true nature of the case.

A localized tympanitic area at this late stage of the disease is suggestive of pneumothorax, tuberculous excavation of the lung, or a peripheral bronchiectasis. In the presence of cavity it is sometimes possible to elicit a cracked-pot sound or one or more of the classial signs of pulmonary cavity, such as Wintrich's or Gerhardt's change of tone described in the first section of this work. A peripheral cavity containing fluid, or a cavity in which the communicating bronchus is occluded by necrotic material, is apt to yield dullness rather than tympany upon percussion. However, it is occasionally possible by repeated percussion blows delivered over the cavity to excite a paroxysm of cough, which effectually expels the contents of the cavity, whereupon percussion of the thorax yields cavernous tympany (Erni's sign).

Upon percussion of the pectoral muscles in chronic ulcerative phthisis a sudden, localized contraction of a muscle is sometimes elicited (myoidema), which is not pathognomonic of phthisis.

Auscultation.—Auscultation of the lungs usually reveals the earliest evidence of the presence of chronic ulcerative phthisis. The sites in which the first alterations of the respiratory murmur are usually encountered are the infraclavicular region, below the inner third of the clavicle, and the interscapular region, above the scapular spine, in the so-called "zone d'alarme" of the French clinicians. The latter area is located by drawing a straight line from the spine of the first dorsal vertebra to the attachment of the trapezius muscle to the spine of the scapula.

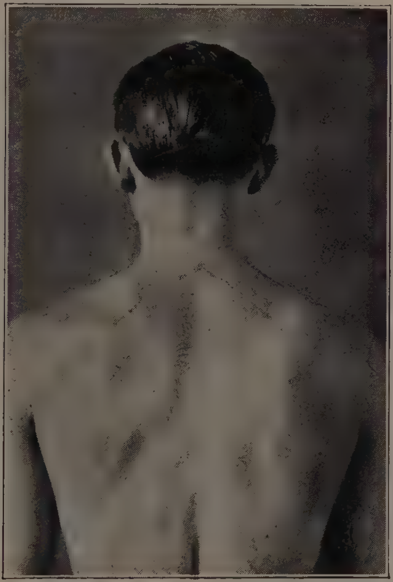

Fig. 286.—Lines indicate areas where râles are usually heard in early stages of chronic ulcerative phthisis.

A circular area two inches in diameter, bisecting this line at its central point, represents the boundaries of the zone of alarm.

The examiner should remember that in dealing with a truly early case the first evidences of the disease are encountered during the inspiratory phase of the respiratory cycle, and that modification of the expiratory phase points to a more advanced stage of chronic phthisis. Frequently the earliest sign of the disease is feeble breathing or suppression of the inspiratory murmur over a limited area of the thorax, usually anteriorly below the inner third of the clavicle, or posteriorly in the interscapular region above the spine of the scapula. However, to possess diagnostic significance such breathing must be distinctly localized, and must be persistent upon successive examinations. It can usually be ascertained that this local finding is attended by a moderate elevation of temperature, associated with acceleration of the pulse rate. This partial suppression of the inspiratory murmur is produced by the formation of peribronchial conglomerate tubercles, causing partial bronchiolar stenosis, or by pleural thickening.

In other cases of early disease the inspiratory murmur is found to be of normal intensity, with the addition of the adventitious element which is recognized as granular respiration. Under these circumstances the examiner obtains the impression that the breath sound is momentarily about to be attended by moist râles, but the râles cannot be identified. When this type of breathing is elicited, it is important that the patient be placed in a posture which is free from muscular tension, as this type of respiration is readily simulated by adventitious muscle sounds.

A comparatively early sign of the disease is the crepitant râle, which at this stage usually requires inspiration after expiration and cough to bring it into evidence. As this râle is dependent upon moisture of the alveolar walls for its production, its presence is at least suggestive that the peribronchial infiltration has penetrated the bronchiolar wall; and it may be attended by expectoration containing tubercle bacilli. As the elicitation of crepitant râles is in no wise pathognomonic of chronic phthisis, crepitation must possess a certain distribution to possess diagnostic significance in this disease. Marginal râles, which are apt to be encountered over the pleural complementary sinus during full inspiration, are to be disregarded. When, however, the râles are encountered in the infraclavicular region or in the interscapular region above the scapular spine, and are attended by limitation of apical resonance or narrowing of Krönig's isthmus upon the same side, they possess potent diagnostic significance.

Interrupted or cog-wheel breathing, while not characteristic of phthisis, is frequently audible over the upper lobes in this disease. Usually the inspiratory murmur alone is involved, but both phases of the respiratory cycle may exhibit interruption. This finding is to be construed in the light of areas of defective resonance, and the clinical features of the individual case. Cog-wheel breathing is encountered in a large number of cases of arrested phthisis, when the pulmonary expansion is limited by pleural adhesions, and in numerous cases of chronic passive congestion of the lungs, in the absence of phthisis.

In the moderately advanced case of chronic ulcerative phthisis the expiratory phase of the respiratory murmur exhibits prolongation over the distribution of a dense infiltration; and it is usually to be observed that the expiratory murmur is unduly harsh. Prolonged expiration may be audible over any portion of the thorax in cases of chronic bronchitis and hypertrophic emphysema, and to possess diagnostic significance in phthisis it must be audible over the superior regions of the thorax. Usually when prolonged expiration is distinctly audible over an upper lobe, subcrepitant râles can be elicited over the area of involvement. As a moderate prolongation of expiration is occasionally a normal finding over the right upper lobe in young individuals with thin chest walls, this type of respiration possesses greater diagnostic significance when it is audible over the left infraclavicular region.

As a rule, with the advent of prolonged expiration over an upper lobe, the respiratory murmur over contiguous portions of the thorax assumes the bronchovesicular type, owing to the dissemination of numerous conglomerate tubercles throughout the air-containing pulmonary parenchyma. Frequently the percussion note over the distribution of this type of respiration will possess a slightly hyperresonant note, due to compensatory emphysema or to local pulmonary relaxation.

In the presence of a more dense pulmonary infiltration, the respiratory murmur becomes distinctly bronchial, frequently with high-pitched tubular expiration. To possess diagnostic significance such respiration must be persistently audible in a more or less restricted area of the thorax, and must be attended by alterations of the percussion sound. Similarly, an extensive peripheral excavation of the lung, with patent bronchial communication, may be attended by bronchial breathing, as may also an area of pulmonary compression above the level of a pleural effusion.

A pleural friction sound is occasionally to be detected above or below the clavicle in the presence of extensive consolidation of an apex. In other instances friction is encountered in the upper interscapular region, or even low in the axillary region.

Vocal resonance is exaggerated over areas of peripheral infiltration, and notably so over superficial cavities with patent bronchial communication. However, the spoken voice is transmitted too loudly to afford definite localizing value in most cases. Of greater service is the transmission of the articular whisper.

While it is true that the articular whisper is audible over the right infraclavicular and upper interscapular region in many normal individuals, in the presence of an apical infiltration or consolidation it assumes a quavering quality which is somewhat similar to egophony, which is of distinct aid in the diagnosis of apical lesions in this disease.

In the late stage of chronic phthisis, presenting the accidents and complications of the disease, the auscultatory findings are quite varied, being represented by the absence of respiration or distant bronchial breathing over a serous effusion or a pneumothorax; by the falling-drop sound, the succussion splash, or the coin test of pyopneumothorax; and by gross alterations of the respiratory sounds over the upper lobes with an extensive distribution of moist râles in these areas. Usually, at this late stage of the disease, the diagnosis may be made before the stethoscope is applied to the thorax.

X-ray.—In chronic ulcerative phthisis the radiograph commonly reveals multiple areas of mottling in the involved area, resulting in a diminution of the transparency of the pulmonary tissues, which is not, however, as dense or as sharply circumscribed as is the shadow cast in lobar pneumonia. On the contrary, in chronic phthisis there are muliple areas of impaired transparency, often superimposed, and frequently not exceeding one-fourth inch in diameter.

In incipient phthisis the mottling is usually circumscribed to the apical or axillary regions, the inferior portions of the lungs remaining clear at this period of the disease.

Partially healed tuberculous lesions, associated with calcareous deposit, give a greater diminution of transparency, producing an altogether more clear-cut picture than that afforded by recent or active tuberculous disease.

The peribronchial lymph glands, when they are involved, cast shadows which must be differentiated from similar shadows due to enlargement of these glands, which are dependent upon the acute infections or syphilis.

Pulmonary cavities, when containing air, are represented by transparent zones, which are usually surrounded by darker zones which correspond to adjacent areas of consolidation of the lung. When cavities are partially filled with fluid a "fluid level" may be made out which changes with the position in which the film is taken.

Fluoroscopic Examination.—The apices of the lung which normally become noticeably less dense during a sharp cough, show

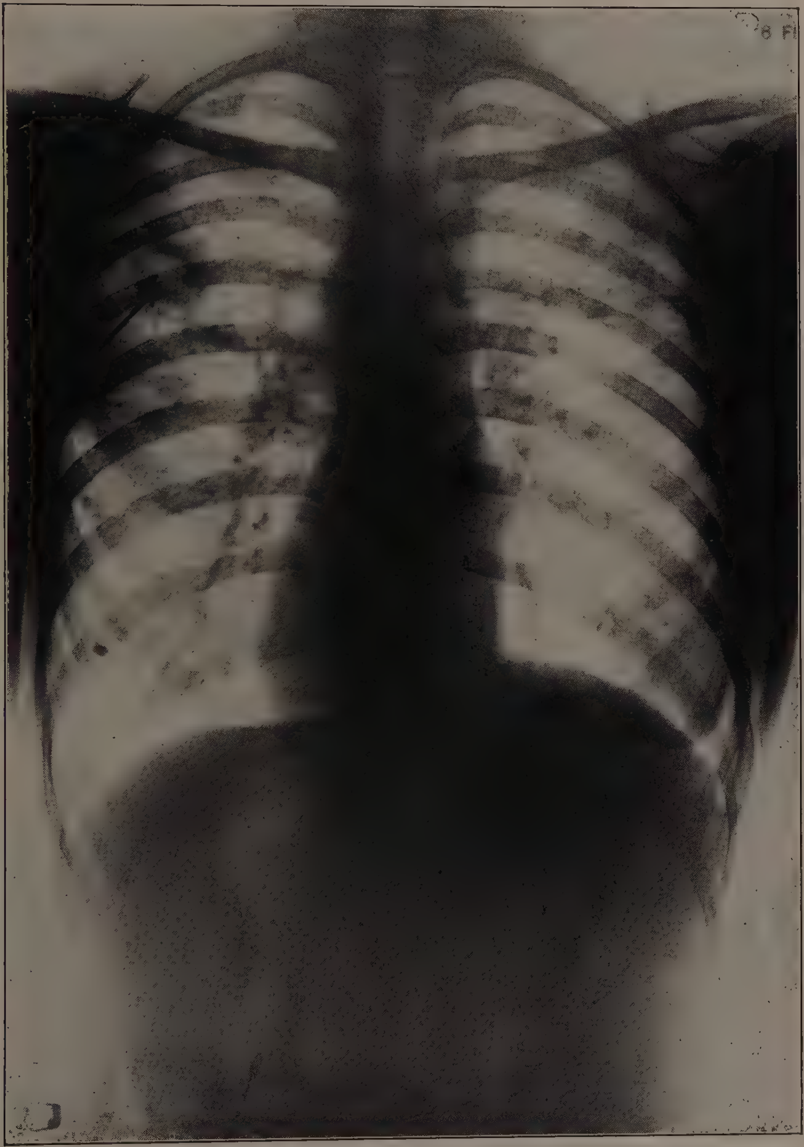

Fig. 287.—Radiograph of advanced tuberculosis with cavity. Arrow indicates the cavity.

little or no change during cough. Diaphragmatic movements may also be restricted or irregular.

Diagnosis.—While the diagnosis of the advanced case of chronic ulcerative phthisis is made with little difficulty, the recognition of truly early phthisis is frequently a difficult matter. In the solution of the latter question the clinician must consider in unison the physical signs, which are frequently meager and inconclusive, and the clinical course and symptoms of the case. Symptoms suggestive of phthisis embrace particularly an evening rise of temperature, which is attended by a pulse rate which is out of proportion to the degree of pyrexia, cough which may be pronounced or a mere clearing of the throat, persistent hoarseness, recent or past hemoptysis, light nocturnal sweating, recent loss of weight, and pains in the chest. It should be borne in mind that 80 per cent of primary idiopathic pleurisy is of tuberculous origin. In certain cases a history of exposure to the disease during childhood may be elicited.

Phthisical fever differs in many respects from febrile elevation due to other causes. The acceleration of the pulse is usually far out of proportion to the degree of temperature, and attacks of paroxysmal tachycardia may be noted. Likewise, the febrile movement of phthisis is not commonly attended by anorexia, and the phthisical patient exhibits an extraordinary ability to work and sleep in the presence of fever. Subnormal morning temperature with a persistent though slight afternoon rise should always demand careful investigation.

A history of hemoptysis is to be very seriously considered in the diagnosis of incipient phthisis. Various workers have encountered this symptom in from 25 to 80 per cent of their cases. It may occur in the form of a single rather profuse hemorrhage, before the physical signs of the disease may be distinguished over the thorax, or it may assume the form of prolonged blood spitting, in a case presenting certain physical signs of chronic phthisis. However, a diagnosis of phthisis is not justified upon this symptom alone, as the raising of blood attends many conditions of other origin, notably diseases of the nose and throat, uncompensated valvular heart disease, pulmonary infarction, syphilis and carcinoma, as well as gastric ulcer or rupture of esophageal varices.

Radiography is of material aid in arriving at a diagnosis in many instances, although in the earliest stage of peribronchial infiltration physical examination of the thorax and the taking of a careful clinical history yield more accurate data than do the x-rays. Similarly, the question of activity or latency of the disease is best determined by physical examination of the patient.

In young children the tuberculin reaction possesses a certain diagnostic value. In the adult subject, however, it is not relied upon by present-day clinicians. Similarly, the complement-fixation test and the autoserum and autourine tests do not afford definite diagnostic information in any considerable proportion of early cases of chronic phthisis.

Routine guinea pig inoculation is of distinct aid in detecting early cases of the disease.

Differential Diagnosis.—Chronic ulcerative phthisis must be differentiated from chronic bronchitis, bronchiectasis, which is not infrequently a concomitant disease, pulmonary carcinoma, syphilis, abscess, or gangrene, streptococcic bronchopneumonia, and pulmonary congestion attending chronic valvular disease of the heart.

Chronic bronchitis is usually a disease of extensive duration, developing in a subject of chronic cardiac or renal disease, who is usually past middle age. The disease commonly extends over a number of years without undue impairment of the health of the individual. The physical signs in chronic bronchitis are generally distributed over both lungs, involving the lower lobes to an extensive degree, in contrast with the more discrete involvement in chronic phthisis. As a rule, when chronic ulcerative phthisis involves the lower lobes to such a degree the patient is in a state of extreme asthenia, and presents characteristic thoracic deformity and changes in more remote portions of the body. It is to be remembered, however, that phthisis in certain aged subjects runs a protracted course, and may extend over a number of years without producing great discomfort. In the end the diagnosis rests upon x-ray studies and the presence or absence of the tubercle bacillus in the sputum.

Bronchiectasis likewise is attended by chronic cough of extensive duration, without undue impairment of the general health. The cough of bronchiectasis is, however, markedly influenced by posture; and, while it is copious, the expectoration is brought up at considerable intervals of time. Bronchiectasis is not usually attended by febrile movement, although there are occasional exceptions to this rule. Similarly, while hemoptysis is less frequent than in chronic phthisis, yet it occurs and sometimes is quite copious.

The distribution of the lesions is different in the two diseases. In bronchiectasis the majority of the dilatations occupy the lower lobes, whereas in phthisis the site of predilection is in the upper lobe or in the apex of the lower lobe. When, as rarely occurs, bronchiectasis involves the upper lobe, it is possible by percussion to elicit normal resonance above the clavicle, a condition which does not obtain in chronic phthisis. Similarly, the depression of the supraclavicular and infraclavicular regions, which are so frequently noted in phthisis, are absent in uncomplicated bronchiectasis, these areas usually exhibiting moderate prominence as a result of compensatory emphysema of the upper lobes. Clubbing of the fingers and signs of pulmonary osteoarthropathy are more frequently noted in bronchiectasis.

Radiographs following the instillation of iodized oil into the bronchial tubes will furnish conclusive evidence of bronchiectasis.

Finally, repeated examinations of the sputum in bronchiectasis fail to reveal the tubercle bacillus, which is usually readily demonstrated in a case of phthisis which is so extensive as to simulate bronchiectasis.

Pulmonary carcinoma is ushered in by an insidious onset and pursues a relatively slow course, with signs and symptoms which simulate chronic ulcerative phthisis in many cases. In the early stage of the disease a positive differentiation can seldom be made by a study of the physical signs, as the dissemination of the neoplastic tissue simulates closely that of chronic phthisis. Gradually, however, pressure symptoms and signs are apt to come into evidence, notably edema of the chest wall, neck or upper extremity, asymmetry of the pulses, and unilateral dilatation of the pupil. Again, in the case of carcinoma, emaciation is not evident so early and does not progress so rapidly as in phthisis, and the acceleration and lability of the pulse are not so constant.

When a tumor of considerable dimensions occupies the upper lobe, percussion over the distribution of the growth yields a note which is duller than that of early phthisis; and, moreover, it is usually possible to demonstrate a resonant or hyperresonant area above the clavicle, which is never present in a tuberculous consolidation of similar extent.

Upon auscultation of the area of dullness, the respiratory murmur is very feeble or is abolished, whereas in phthisis with such an extensive area of consolidation the murmur is commonly bronchial, with superadded moist râles throughout the dull area. Frequently, in the case of a carcinoma of extensive dimensions, a large bronchus is occluded, resulting in atelectasis and dullness with absence of breath sounds over an extensive area of the thorax.

A valuable differential sign is the position of the cardiac impulse, as well as that of the trachea. Whereas in the case of phthisis of extensive distribution, these structures are commonly drawn toward the side of the major lesion, in the case of carcinoma they are usually displaced in the opposite direction.

When an extensive tumor develops in the lower lobe, it is likely to simulate in its physical manifestations the effusion of fluid into the pleural cavity. As a matter of fact, both chronic phthisis and pulmonary carcinoma are attended by pleural effusion in many cases. However, in the case of carcinoma the effusion is usually much more abundant, filling a large portion of the pleural cavity. Under these circumstances the upper zone of Skodaic resonance which is usually to be demonstrated above the level of a tuberculous effusion, is absent in the effusion due to pulmonary carcinoma.

During all periods of the disease radiography of the thorax is of distinct aid in differential diagnosis. Tumors, as a rule, show greater density and more circumscribed borders.

Pulmonary syphilis in certain instances simulates chronic ulcerative phthisis; but it more frequently approaches the clinical picture of the fibroid form of the disease. In syphilis the lesions are usually grouped about the root or middle lobe of the lung, leaving a broad band of resonance above and below the central area of impaired resonance. The course of the disease is seldom progressive, and it does not greatly incapacitate the patient. The specific tests for syphilis frequently are of aid in arriving at a differential diagnosis.

Pulmonary abscess is a disease of acute onset following aspiration pneumonia or arising as a result of septic embolism of the pulmonary artery. Its course is rapid and stormy, with fever, cough which is attended by foul expectoration, sweats, emaciation, and occasionally hemoptysis. The site of the lesion in this case usually but not invariably occupies a lower lobe. Even in the cases in which the upper lobe is involved the supraclavicular regions are usually clearly resonant, as the lesion is located below the level of the clavicle.

The radiograph may show a more or less extensive excavation, usually with thin walls. A fluid level can also be demonstrated at times. Instillation of iodized oil may aid in such a demonstration.

Pulmonary gangrene is attended by the expectoration of large quantities of foul sputum, containing acid-fast bodies which bear some morphologic resemblance to the tubercle bacillus. The history of an acute onset following an aspiration pneumonia, the character of the sputum, and the localization by choice in the lower lobe are of aid in differential diagnosis. The expectoration in gangrene of the lung is exceedingly foul, whereas that of chronic phthisis is seldom fetid.

Certain cases of *streptococcic bronchopneumonia* pursue a subacute or chronic course, which may simulate for a time the picture of chronic ulcerative phthisis. In the majority of cases, however, the lesions are located in a lower lobe and may involve a considerable portion of a lobe. In this event, the percussion note over the upper lobe is not impaired. In the cases in which the disease occupies an upper lobe, the respiratory murmur is commonly bronchovesicular, and moist râles are audible over the apex; but examination of the sputum reveals various strains of the streptococcus.

Valvular heart disease, notably mitral stenosis, is sometimes attended by pulmonary stasis, which leads to brown induration of the lung, which is characterized by expectoration which is frequently blood-tinged, with mild elevations of temperature and moderate loss of weight. Dyspnea is frequently more severe and more persistent than is the case in chronic phthisis. Examination of the bases posteriorly serves to reveal widely distributed moist râles, and auscultation of the precordia reveals the cardinal signs of a valvular lesion.

Malaria under certain circumtances may simulate the course of early phthisis, particularly the estivoautumnal type of infection. Examination of the cytology of the blood serves to clear up the diagnosis in such cases.

Actinomycosis resembles in its clinical course chronic ulcerative phthisis in numerous instances of the disease. Examination of the sputum, however, reveals the presence of characteristic actinomyces granules, which contain the streptothrix actinomyces.

FIBROID PHTHISIS

Clinical Pathology.—In this form of chronic phthisis the predominant change is the formation of an excessive amount of fibrous connective tissue, involving different areas of the lung, and tending toward the production of numerous bronchiectases. The causative factors of this productive form of phthisis have not been definitely determined; but pathologic studies suggest that the fibrous overgrowth is to be attributed to stimulation of the tissues by the tubercle bacillus, as typical giant cells can be demonstrated in the midst of the fibrous lesions. In this respect the pulmonary fibrosis attending the disease differs essentially from the cirrhosis of the lung which occurs in chronic interstitial pneumonia.

The combination of syphilis and phthisis has been urged as a cause of fibroid phthisis; but, while the two diseases sometimes are associated, the specific tests for syphilis are negative in the vast majority of cases of fibroid phthisis. It has been assumed, but not proved, that this form of the disease is caused by bovine bacilli or by human bacilli which have become attenuated in their virulence. It has also been suggested that the occasional demonstration of Much's granules in the fibrous lesions points to the fact that fibroid phthisis is caused by bacilli which have lost their acid-fast properties. A good many cases have been noted in persons engaged in occupations which occasion the inhalation of irritant dusts, suggesting an association of the etiologic factors which are productive of pneumoconiosis.

Three types of fibroid phthisis are distinguished: namely, the *simple fibroid* form, the *emphysematous* form, and the *pleurogenous* form, of the disease.

In the *simple fibroid* form a peribronchial fibrosis with its inception in an apex of the lung tends to progress slowly over a number of years, with periods of remission, during which symptoms disappear and the patient is apparently "cured" of his disease. From the point of inception, the extension of the disease is backward and downward; and the disease gradually involves a lobe or an entire lung. During the later stages of the disease, caseation occurs, with the production of pulmonary cavities. In this type of chronic phthisis bronchiectatic cavities, arising as a result of the traction upon the bronchi of fibrous bands, are encountered with greater frequency than is the case in chronic ulcerative phthisis.

The disease is attended by mucopurulent expectoration in which tubercle bacilli are usually demonstrable. Hemoptysis is frequently noted, and fatal hemorrhage is not infrequent.

The pleura is constantly thickened over the site of the disease, but effusion into the pleural sac is not frequently encountered.

In the *emphysematous* form of fibroid phthisis the area of pulmonary fibrosis is commonly limited to an upper lobe in the region of the apex, while the adjacent and more distant portions become compensatorily emphysematous. Over the distribution of the fibrosis the pleura is thickened, frequently to an extreme degree; and the apical portion of the lung is often bound to the parietal pleura by dense bands, immobilizing the superior portion of the lung. The subject of the disease is liable to persistent dyspnea, which is more pronounced than in the ulcerative type of chronic phthisis, while the thoracic contour tends to approach that of hyperthropic emphysema.

With the further progress of the disease, cavitation in the area of fibrosis is the rule, and bronchiectasis involving the upper lobe is frequently noted. Tubercle bacilli are usually to be demonstrated in the sputum, and hemoptysis or frank hemorrhage is not infrequent.

The *pleurogenous* form of fibroid phthisis follows in the train of an attack of serofibrinous pleurisy, which represented tuberculosis of the pleura. In this form of the disease the investing pleura is greatly thickened, sometimes attaining a thickness of two centimeters; and, over the site of the lesion, which in this instance not infrequently occupies a lower lobe, the two pleuræ are occasionally firmly adherent, resulting in immobilization or even retraction of the lower portion of the thoracic wall during inspiration.

From the area of pleural thickening, bands of fibrous tissue penetrate the depths of the lung, and are a productive source of bronchiectasis and bronchial occlusion. Excavation of the involved area of the lung eventually supervenes, and the resulting cavities are frequently surrounded by dense fibrous capsules.

All forms of fibroid phthisis are attended by a greater degree of bronchitis than is the chronic ulcerative form of the disease. Similarly, dyspnea is a more striking feature of fibroid phthisis.

Physical Signs.—*Inspection.*—The physical manifestations of fibroid phthisis vary with the type which the individual case assumes, and with the duration of the disease. In many early cases the thoracic contour is normal for the individual.

In the simple fibroid form of the disease, for a long time the only sign may be moderate dyspnea upon exertion. With the further progress of the disease, however, it is usually possible to detect lagging of the affected apex upon inspection of the thorax from a position behind the patient. Likewise, it is to be noted that the supraclavicular fossæ are of unequal depth when the thorax is in repose. In other instances repeated attacks of hemoptysis are the sole manifestations of the disease.

In the emphysematous form of fibroid phthisis the thoracic contour is often suggestive of hypertrophic emphysema, although there are numerous exceptions to this rule. A more frequent finding upon inspection is a moderate flattening of the superior thoracic region, with a tendency of the costal margins to flare outward to a variable degree. The shoulder upon the side of the original lesion may present moderate dropping, and lagging of the affected side is usually perceptible. In this form of the disease dyspnea is a prominent feature of the case throughout its course, and local cyanosis is occasionally noted with the patient in repose.

In the pleurogenous form of the disease the expansion of the inferior and lateral regions of the thorax upon the affected side is very frequently defective; and, in the presence of extensive fibrosis of the lung, there may be inspiratory retraction of the lower interspaces. The opposite side of the thorax, on the contrary, is observed to be compensatorily voluminous, and mensuration reveals a distinct disproportion between the two sides of the thorax.

When the fibrosis predominates in the left lung, there is frequently a broad visible impulse along the left sternal border, caused by retraction of the lingula of the left lung which normally overlaps the heart in this region. In the case of extensive disease of the right lung, on the contrary, it is not uncommon to find the cardiac impulse invisible and impalpable, owing to displacement of the heart toward the right.

Clubbing of the fingers, incurvation of the nails, and signs of pulmonary osteoarthropathy are more commonly noted in fibroid phthisis than in other forms of this disease.

Palpation.—In fibroid phthisis palpation of the trachea usually reveals a lateral deflection of this structure toward the side of greater fibrosis. In cases of apical fibrosis, gentle palpation of the supraclavicular regions during inspiration occasionally serves to reveal lagging of the affected apex. Likewise, in the pleurogenous form of the disease, associated with pleural adhesions, the lateral mobility of the chest wall is restricted upon the side of the disease.

Vocal fremitus is apt to exhibit a moderate exaggeration over the infraclavicular and upper interscapular region in the presence of extensive fibrosis of an upper lobe; but frequently the associated pleural thickening serves to obscure the vocal vibrations which are generated in the laryngeal cavity during phonation. On the whole, definite conclusions cannot be based upon variations of vocal fremitus in this type of phthisis. Similar variations are sometimes encountered over the area of fibrosis in cases of pleurogenous origin; but the findings are seldom in any wise conclusive.

Percussion.—In the simple fibroid and emphysematous forms of the disease percussion of the thorax reveals impaired resonance over one or both infraclavicular regions. Likewise it is usually possible to demonstrate a distinct narrowing of Krönig's isthmus upon the side of the disease. In cases which are attended by a considerable grade of compensatory emphysema, moderate hyperresonance can be elicited over the lower regions of the thorax, and the pleximeter finger frequently appreciates a sense of increased resistance. Similarly, under these circumstances it is usually to be noted that the inferior border of the lung occupies an abnormally low level, and that its inspiratory excursion is definitely restricted.

In the pleurogenous form of the disease, over the distribution of an area of extensive pleural thickening with adhesions, the percussion note is impaired, or is frankly flat, simulating the findings in pleurisy with effusion. Percussion of the opposite side of the thorax usually yields a mildly hyperresonant note, due to compensatory emphysema of the subjacent lung. Delimitation of the pulmonary borders not infrequently reveals an elevation of the inferior border of the lung upon the side of the disease, with a coincident extension of the lower border of resonance upon the opposite side of the thorax.

Auscultation.—In the simple fibroid and emphysematous forms of fibroid phthisis auscultation of the thorax is apt to reveal the ordinary signs of an apical consolidation in the infraclavicular and upper interscapular regions; namely, prolongation of expiration, bronchovesicular or frankly bronchial breath sounds, intermingled with distant or loud and consonating râles. In other instances, associated with little bronchial inflammation and considerable pleural thickening, the respiratory sounds are diminished in intensity or are abolished over the distribution of the upper lobe. Not infrequently in these cases a pleural friction sound is audible during full inspiration. Similarly, in the presence of excavation of the upper lobe, the veiled puff may be audible toward the completion of inspiration.

In the pleurogenous form of the disease auscultation of the lower lateral thorax may reveal a distant respiratory murmur over an area of pleural thickening, or there may be distant bronchial breathing with râles over an area of fibrosis. As a rule, auscultation of the affected side serves to reveal signs of an old tuberculous focus in the upper lobe of the lung.

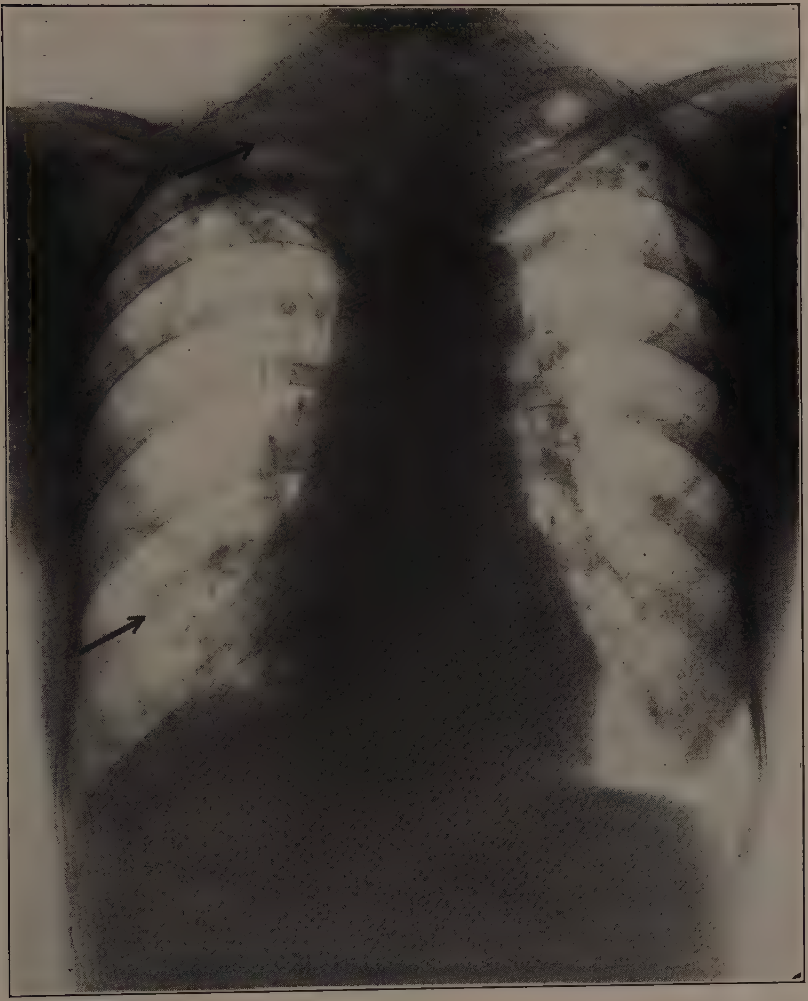

Fig. 288.—Radiograph of fibroid tuberculosis. Arrows indicate areas of consolidation and fibrosis.

X-ray.—The pulmonary apices usually show increased density. The lung structure is obscured by irregular areas of density due to thickened pleura. Hilus and bronchial shadows are increased. The diaphragm may be irregular due to adhesions between the pulmonary and diaphragmatic pleura. The pericardium may also be adherent to the pulmonary pleura.

Diagnosis.—During the latter stages of the disease the clinical picture of fibroid phthisis resembles in many of its essential points that of chronic ulcerative phthisis. Earlier cases frequently present little more than a persistent dyspnea which appears to be out of proportion to the physical signs which can be elicited. However, examination of the sputum may reveal tubercle bacilli, if a persistent search is made for them.

The extensive bronchial involvement which attends certain cases of fibroid phthisis is apt to suggest the diagnosis of chronic bronchitis; but hemoptysis is frequent in fibroid phthisis, while it is rare in chronic bronchitis. During a casual examination the clubbing of the fingers and signs of pulmonary osteoarthropathy are apt to cause confusion with bronchiectasis.

Radiography of the thorax will frequently serve to clear up the diagnosis in early cases. The bilateral distribution of the disease is likewise more suggestive of phthisis than of a non-specific pulmonary fibrosis. The final test is the presence or absence of tubercle bacilli in the sputum.

PULMONARY SYPHILIS

Clinical Pathology.—Pulmonary syphilis occurs in two forms; namely, as *congenital syphilis,* and as *acquired syphilis* of the lung.

Congenital syphilis of the lung was first described by Virchow as "pneumonia alba." The lung in congenital syphilis is uniformly enlarged, showing upon its external surface indentations corresponding to the ribs with which it is in contact. The lung is white or slightly tinged with yellow, firm, and upon section the cut surface resembles macroscopically a section of pancreatic tissue, a condition to which Lorain and Robin applied the term "pancreatization."

Microscopically the interalveolar septa show an overgrowth of fibrous connective tissue, leading to thickening of the alveolar walls, the alveolar spaces being smaller than normal and densely packed with desquamated epithelial cells, cellular detritus, and fat. Hoffman called attention to a thickening of the vascular walls in the interalveolar septa, analogous to that which occurs in syphilitic fetal tissue elsewhere, the vessels often presenting, as well, evidences of hyaline degeneration.

The lesions of acquired syphilis of the lung may assume three types; namely, gummata, interstitial sclerosis analogous to chronic interstitial pneumonia from other causes, and syphilitic bronchopneumonia.

The gummata are situated deeply near the root of the lung, varying in size from a hazelnut to a hen's egg or larger. They are prone to soften and to break into a bronchus or to undergo sclerosis and by traction lead to bronchiectatic dilatations. Gummata are the rarest of syphilitic lesions of the lungs. Wagner and Henop described gummata in both the upper and lower lobes of the lung, situated principally toward the root, usually containing in their center a dilated bronchus with chronically inflamed mucous membrane. The lung intervening between the gummata was partially crepitant, while the apices and anterior borders of the lungs were in a state of compensatory emphysema.

The interstitial sclerosis attending acquired syphilis of the lung has its inception near the root of the lung, and extends thence in various directions between the lobules of the lung. The patches of insular sclerosis often extend in all directions from gummata situated near the pulmonary root, dividing the lung into a number of artificial subdivisions. Traction diverticula are formed, which lead to bronchiectases.

The bronchopneumonia of syphilis does not differ essentially from bronchopneumonia of other causes, Pavloff describing a bilateral, syphilitic bronchopneumonia with patches of consolidation interspersed between areas of normal tissue in both lungs. The alveoli are filled with desquamated epithelial cells, with a variable number of erythrocytes and leucocytes, the pneumonic patches being dull slate-colored upon section.

Acquired syphilis of the lung is not a common affection; and, indeed, certain clinicians have denied its existence as a clinical entity.

The disease pursues a chronic course, without greatly influencing the health of the subject. It is attended by rather abundant mucopurulent expectoration, which is not infrequently sanguineous. Indefinite periods of mild pyrexia are occasionally observed, usually associated with an exaggeration of the pulmonary signs of the disease.

Physical Signs.—The physical signs of acquired pulmonary syphilis are not characteristic, the clinical picture closely simulating that of chronic interstitial pneumonia, chronic ulcerative phthisis, or ordinary bronchopneumonia in different instances. However, there are certain localizations of the principal signs emanating from pulmonary syphilis which are suggestive of lues. Thus the lesions are usually situated near the hilus of the lung, gummata and sclerosis in this situation affording dullness upon percussion and bronchovesicular breath sounds along the lateral sternal borders, the signs diminishing in intensity as the examiner progresses toward the periphery of the lung. Grandidier lays special emphasis upon the localization of the physical signs in these regions of the thorax, while Pankritius points out the importance of similar signs in the interscapular region at the same level.

The x-ray may demonstrate densities at the periphery of the lung but on the whole there are no typical x-ray findings.

Diagnosis.—Congenital syphilis of the iung must in certain instances be differentiated from atelectasis, which it closely simulates; but there is usually sufficient evidence of congenital syphilis upon the exterior of the body to render the differential diagnosis easy.

Acquired syphilis, with its irregular fever and chronic cough, which is frequently attended by hemoptysis, simulates rather closely the picture of chronic ulcerative phthisis. The absence of physical signs over the apices and the predominance of signs over the hilus region, however, serve to suggest syphilis rather than phthisis. Similarly, the sputum upon repeated examinations remains free from tubercle bacilli. There are apt to be evidences upon the body of former syphilitic lesions, and the specific tests for lues are frequently positive.

PNEUMONOCONIOSIS

Clinical Pathology.—Pneumonoconiosis is a chronic induration of the lungs due to the inhalation of various dusts and mineral particles. Depending upon the nature of the exciting irritant, the disease is subdivided into several types: *siderosis,* from the inhalation of iron filings; *chalicosis,* from the inhalation of stone particles; and *anthracosis,* from the inhalation of coal dust. Similar pulmonary changes ensue upon the prolonged inhalation of the fibers of wool, flax, cotton, tobacco, or particles of glass, bone, or horn, in the course of an occupation.

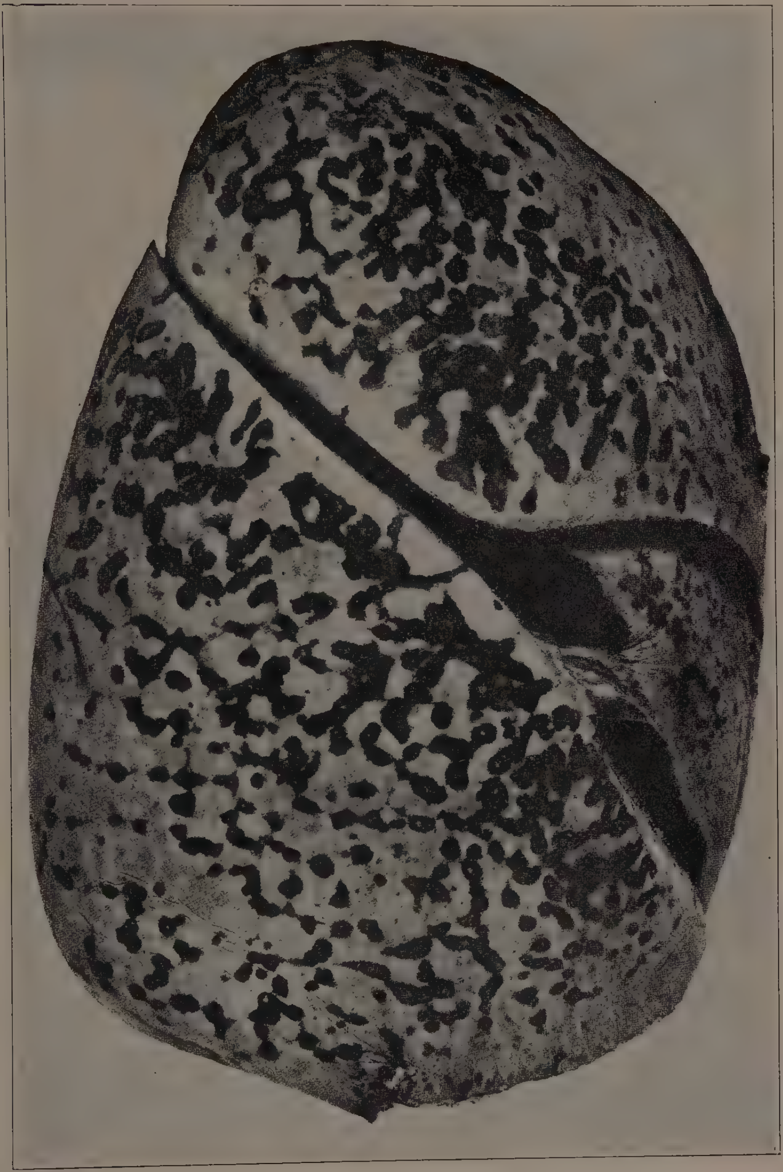

Fig. 289.—Anthracosis. (From Delafield and Prudden.)

Anthracosis, or "coal miner's disease," arises as the result of the prolonged inhalation of fine particles of coal. The minute amounts of this dust which are usually inhaled by persons who do not pursue an occupation which habitually brings them into contact with coal dust are absorbed by the leucocytes which are present upon the surfaces of the respiratory passages; and they are carried upward by the action of the ciliated epithelium of the respiratory tract, and are expectorated. When, however, the dust is inhaled in larger amount, some of the coal particles penetrate the bronchial mucous membrane and find lodgment in the subjacent connective tissue layer, or enter the lymph stream and are conveyed to the smaller lymphatic glands around the blood vessels, the bronchi, the pleura, or in the mediastinum. The lungs of all dwellers in cities are moderately pigmented from the inhalation of coal dust and soot, while the lungs of persons who have lived all their days in the open country, remote from large manufacturing industries, are frequently pink in color and free from this pigmentation.

When soot or coal dust is inhaled in excessive quantities, as in the case of the coal miner, a portion of it penetrates to the ultimate ramifications of the bronchioles and to the pulmonary alveoli. In the lungs which are the seat of this extreme grade of anthracosis the organs are distinctly black. The irritation of the fine grains of dust in the interstices of the pulmonary tissues excites the proliferation of fibrous connective tissue, resulting in fibrosis in insular foci. Upon section these fibrosed areas are hard to the touch and they exude a black fluid. Diffuse induration of an entire lung, or of the greater portion of a lung, is commonly encountered at autopsy.

The bronchial and mediastinal lymphatic glands share in the general induration, and they are frequently the seat of periadenitis by virtue of which they adhere to the adjacent large vessels, and by rupture into the same they distribute the pigmentation to various organs of the body, as the liver, spleen, kidneys, and mesenteric lymphatic glands.

Bronchial perilymphadenitis may lead to the formation of adhesions between the glands and the pericardium, producing a chronic mediastinopericarditis. Adhesions are apt to form between these glands and the esophagus, and lead to the formation of esophageal diverticulum. Similarly adhesions are prone to form between the mesenteric glands and the aorta, with subsequent erosion of that vessel with fatal hemorrhage. Adhesive bands may also constrict the trachea or adjacent blood vessels, producing tracheal stenosis with stridulous respiration, or vascular constriction with the production of murmurs. Aspiration pneumonia may result if a gland erodes a bronchus and discharges its contents by that avenue.

Moreover, the chronically inflamed glands may produce vagus or recurrent laryngeal nerve paralysis by pressure upon these nerves.

Chalicosis, due to the inhalation of the fine dust of alumina, quartz, or sandstone, is known as ''stone-cutter's phthisis,'' millstone maker's phthisis,'' ''grinders rot,'' or ''potter's asthma.'' Chalicosis produces a greater degree of induration of the lung than does any other form of pneumonoconiosis.

Siderosis is a fibrosis of the lung due to the inhalation of iron filings or iron dust. The changes in the lungs and the mediastinal and bronchial glands are similar to those which accompany anthracosis; but the induration attains a greater degree than it does in anthracosis.

The areas of insular sclerosis which are formed throughout the lungs, in certain cases undergo softening and form pulmonary cavities. In other instances the softening is due to subsequent infection with the tubercle bacillus; although, as a rule, the pneumonoconiotic lung does not appear to prove a fertile field for the ravages of the tubercle bacillus. As a result of the chronic inflammation which is induced by the continual aspiration of the irritant dusts, the bronchial tubes show a chronic bronchitis, which is followed in many cases by emphysema, while the lung slowly undergoes a slow, insular sclerosis.

Physical Signs.—The physical manifestations of pneumonoconiosis are quite variable, depending as they do upon the character of the irritant which is provocative of the disease, the duration of the individual case, the combination of the disease with chronic bronchitis or emphysema, and the degree of insular or general sclerosis of the pulmonary tissues which is induced.

Inspection.—In an early case of pneumonoconiosis, while the subject is in repose, no signs are in evidence which serve to suggest a diagnosis of this condition. It will usually be found that the patient is subject to dyspnea upon moderate exertion, which is the predominant finding in this early stage of the disease. In the advanced case, however, it is usually to be observed that the expansion of the two sides of the thorax is unequal, while the total thoracic expansion exhibits a bilateral restriction. In different cases this deficiency may prove most obvious over the superior or the inferior portions of the thorax.

If the left lung has undergone a considerable degree of sclerosis, with retraction of its anterior border, the cardiac impulse is to be noted as a broad pulsation in the third, fourth, and fifth interspaces between the left sternal border and the mammary line. In certain cases the apex-beat is displaced far to the left of the latter line. Similarly, in the presence of extensive retrogressive changes in the right lung, the cardiac impulse may be invisible, as the cardiac apex is displaced behind the sternum.

The emphysematous type of the thorax is occasionally encountered in subjects of this disease; but, as a rule, the changes in the lungs occur during adult life, at which time the thorax is too rigid to be influenced by the emphysematous changes in the lungs. In cases of pneumonoconiosis which are associated with phthisis, there are no characteristic physical signs which indicate this fact. The detection of the tubercle bacillus in the sputum is the deciding factor in this event.

Early in the course of the disease the sputum is scanty and mucoid, sometimes containing particles of the provocative agent of the disease. In the later stages, which may be associated with chronic bronchitis and pulmonary excavation, the sputum is abundant, thin, and occasionally sanguineous.

Palpation.—Finger-tip palpation of the superior and lateral regions of the thorax is of service in the detection of minor deficiencies of thoracic expansion in this disease. Similarly, palpation serves to localize more accurately the site of the cardiac impulse, which in the presence of advanced disease may be displaced far from its normal site.

Vocal fremitus may be increased or diminished in intensity over different areas of the thorax, depending upon the state of the lungs and the investing pleura. Deductions based upon variations in vocal fremitus lead to erroneous conclusions in numerous instances of this disease.

Percussion.—Percussion of the thorax yields no characteristic findings in pneumonoconiosis. In cases with a tendency toward hypertrophic emphysema the percussion note is slightly hyperresonant upon both sides of the thorax. In cases with insular sclerosis, on the contrary, areas of defective resonance are apt to be encountered over the upper lobes, the hilus regions, or the bases, findings which are in no wise characteristic of pneumonoconiosis. Cases which are associated with large bronchiectases sometimes yield a moderately tympanitic percussion note in the infraclavicular and upper axillary regions.

Auscultation.—The auscultatory findings in pneumonoconiosis are variable and indefinite, resembling markedly those obtained in chronic bronchitis during the early stages of the disease, and closely simulating those of chronic phthisis in later cases. When, as a result of exposure to irritant dusts, the chronic bronchitis of the disease becomes established, moist râles usually become audible over both sides of the thorax, with no characteristic distribution. There are instances of the disease attended by insular sclerosis of the upper lobe, however, in which the signs are largely confined to the infraclavicular region, closely simulating in this respect the findings of early phthisis. In these cases the respiratory murmur is frequently bronchovesicular and occasionally frankly bronchial in character.

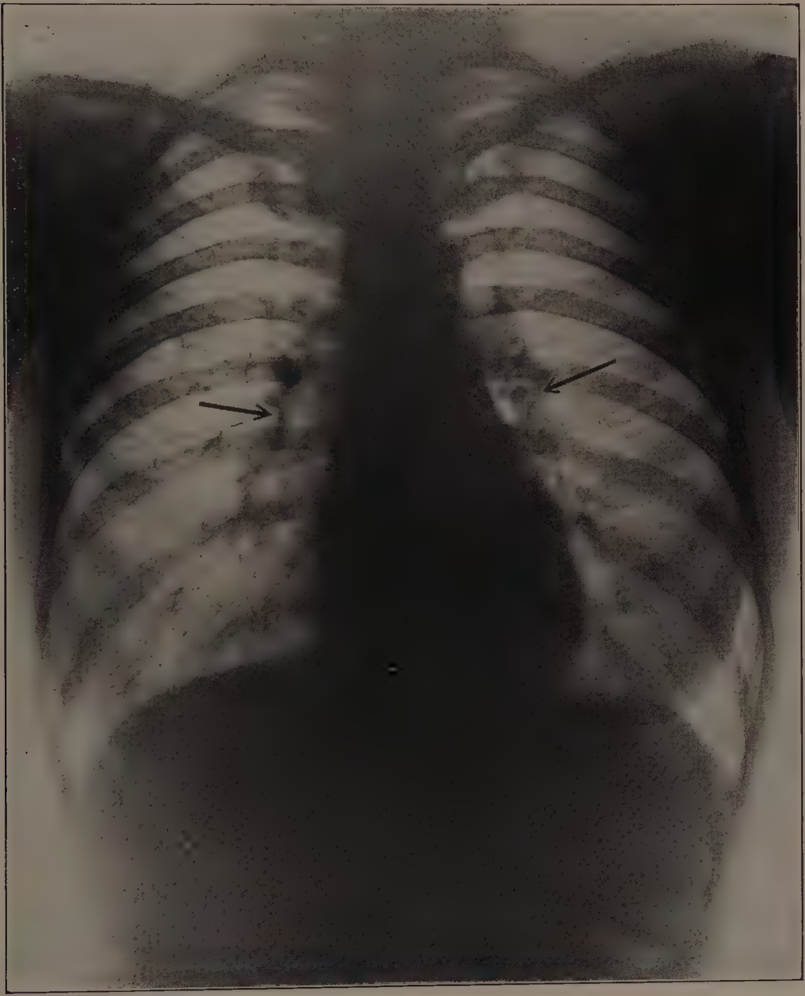

Fig. 290.—Radiograph of anthracosis. Arrows indicate density caused by deposits of carbon.

With the advent of pulmonary excavation, in late stages of the disease, the signs of cavity become apparent. Cases of extensive peripheral distribution are frequently attended by a pleural friction sound.

X-ray.—The hilus shadows are markedly increased and of unusual density. Areas of increased density may be noted along the course of the bronchi and are especially noted at the bifurcations.

Diagnosis.—During an early stage of the disease a diagnosis of pneumonoconiosis can seldom be made in the absence of radiographs of the chest. Occasionally particles of mineral dusts can be detected in the sputum, a finding which is of assistance when it is available. As a rule, the original diagnosis in such cases is chronic bronchitis in the presence of bilateral disease, or incipient phthisis when an apical sclerosis predominates the clinical picture. The history of exposure to irritant dusts usually furnishes a clue to the cause of the bronchial signs and symptoms.

A coincident chronic phthisis in a subject of pneumonoconiosis cannot be diagnosed by the physical signs, but must rest upon the demonstration of the tubercle bacillus in the sputum.

ATELECTASIS

Clinical Pathology.—Imperfect expansion of the lung or partial collapse of a lung which has become fully expanded may be congenital or acquired. In the congenital form of atelectasis the lung has never attained its proper degree of expansion; while in the acquired form, which is a disease of later life, there occurs from various causes a partial collapse of the lung.

Congenital atelectasis is a disease of the newly born, developing usually as a result of insufficient inflation of the lung due to the aspiration of meconium or mucus during parturition, or from abnormal weakness of the respiratory muscles subsequent to birth. In atelectatic children who survive, the anterior borders and the apices of the lungs are partially inflated, while the central and lower portions of the lungs are brownish-red, vascular, and fail to crepitate upon manipulation.

As the child gains strength the anterior and upper portions of the lungs become emphysematous from compensatory emphysema, while the deeper portions are very tardy in attaining their proper degree of expansion. Congenital atelectasis is a bilateral disease, affecting both lungs, and usually to a similar degree. It is probable that the central portions of the lungs of atelectatic children never attain to full inflation, as the tendency is rather for secondary changes to occur, which lead to sclerosis and contraction of the deeper portion of the lung near the pulmonary root.

Acquired atelectasis is frequently the result of compression of the lung by a pleural effusion, a tumor, aneurysm, or deformity of the thoracic wall. The most fruitful source of the disease, however, is bronchial obstruction from foreign bodies, or external pressure from a pulmonary tumor or aortic aneurysm. Similarly, in the course of capillary bronchitis or bronchopneumonia the terminal bronchioles are obstructed by inflammatory exudate, leading to circumscribed areas of atelectasis. Conditions of great debility, the result of malnutrition or lying long in the recumbent posture with an exhausting disease, occasionally induce areas of circumscribed atelectasis.

The atelectatic area of the lung is darker than the adjacent tissues, is depressed below the surface of adjacent areas of the lung, and is usually distributed in a number of areas corresponding to lobules of the lung. Upon section the areas are usually dry, but they may be moist from the presence of chronic passive congestion.

Physical Signs.—The physical signs of atelectasis are influenced and modified by the primary manifestations of the disease or condition which has been the occasion of the atelectatic state of the pulmonary tissues. The signs also vary in intensity with the volume of lung involved in the atelectasis and the condition of the undiseased portion of the bronchopulmonary system. It is obvious that the physical signs arising from collapse of an entire lobe or an entire lung will differ markedly in distribution and degree from those which are referable to a few scattered areas of pulmonary collapse.

Inspection.—Extensive atelectasis, involving a considerable area of a lung, gives rise to dyspnea and occasionally to cyanosis, with inspiratory retraction of the lower intercostal spaces and the epigastrium. In congenital atelectasis the child suffers with repeated suffocative attacks during which immediate dissolution seems to be imminent. The expansion of the thorax is materially diminished in extensive atelectasis.

Palpation.—Vocal fremitus over the area of disease may be diminished, absent, or exaggerated, depending upon the state of the pulmonary parenchyma. Collapsed, toneless pulmonary tissue fails to conduct the vocal vibrations with the normal intensity; and if a main bronchus is completely obstructed, there is absence of vocal fremitus over the distribution of the bronchus. However, when secondary changes have been established in a case of atelectasis with partial bronchostenosis, the fremitus is transmitted by the consolidated or sclerotic tissues with undue intensity to the thoracic wall. Pleural friction fremitus is occasionally demonstrable, due to involvement of the pleura over the atelectatic area.

Percussion.—The dullness which would be produced by small patches of atelectasis is effectually masked by the hyperresonance of the adjacent emphysematous areas. In order to afford dullness upon percussion an area of atelectasis must be large and situated in the peripheral portion of the lung. A patch which directly overlies a large bronchus yields the tympany of the bronchus upon forcible percussion. Deeply seated areas of atelectasis fail to yield dullness, owing to the intervention of the normal crepitant tissues between the area of disease and the chest wall.

Auscultation.—The vesicular murmur is feeble or abolished over an area of atelectasis, unless the area of collapse overlies a large bronchus, in which event the murmur is bronchovesicular or frankly bronchial. In the presence of nonextensive areas of collapse the only auscultatory sign may be the presence of a few crepitant râles upon full inspiration. This holds true of the cases of atelectasis which develop in the dependent portions of the lungs in patients who have been long in the recumbent posture with exhaustive disease. In cases of extensive atelectasis the second sound of the heart is accentuated at the pulmonary area.

X-ray.—Areas of atelectasis being airless react to the x-ray as do consolidations.

Diagnosis.—The diagnosis of pulmonary atelectasis is frequently for a time difficult or impossible. The finding of respiratory embarrassment and the location of some adequate causative lesion such as bronchial obstruction or pulmonary compression is suggestive. The physical signs are seldom definite and distinctive, as small areas of collapse have their physical signs effectually masked by the emphysematous state of the surrounding lung. Moreover, as a larger area of collapse is apt to overlie a large bronchus and to have its tympanitic note engrafted upon the dullness of the atelectasis, a pulmonary cavity is apt to be suspected where none exists. The fact that the physical signs have a tendency to improve and to regress with changes of posture and with deep inflation of the lungs is suggestive of atelectasis.

MASSIVE COLLAPSE OF THE LUNG

Massive collapse of the lung may result from any of the following causes: following operations in the upper abdomen, sufficient interference with diaphragmatic movements and abdominal musculature may occur to produce collapse of a considerable area or even an entire lung.

Diaphragmatic paralysis may produce the same effect, as may also pneumothorax. Obstruction of a primary bronchus by a foreign body may cause a collapse of that portion of the lung which it supplies. As a result of one or more of the above factors, a varying portion of the lung becomes collapsed. The collapsed lung appears smaller, darker in color, and of a leathery feel.

The Physical Signs.—If collapse is sudden and complete, symptoms of shock may develop, followed by temperature of 100 to 102°. The affected side seems shrunken, the intercostal spaces are depressed, Litten's sign is diminished or absent. The apex impulse is displaced toward the affected side. The unaffected side is usually apparently enlarged and overactive.

Palpation.—The tactile fremitus is increased over the affected area unless the bronchus is occluded, and diminished over the unaffected area. The displacement of the apex impulse is confirmed.

Percussion.—Dullness over the affected area with increased resonance over the uninvolved portion. The heart dullness is displaced toward the affected side.

Auscultation.—Bronchial or bronchovesicular breath sounds depending on the degree of collapse. If the bronchi are occluded or kinked, breath sounds may be absent. Both whispered and spoken voice sounds are increased unless, as noted above, the bronchi are occluded, in which event they are diminished or absent. A few moist râles may be noted.

The radiograph shows a dense shadow over the affected area, with displacement of the heart and mediastinal contents toward the affected side. The diaphragm is elevated on the affected side.

Fluoroscopic examination will demonstrate decreased diaphragmatic movements on the involved side with increased movements on the unaffected side.

The Diagnosis.—Many cases diagnosed postoperative pneumonia are probably massive collapse of the lung. The shrinking of the affected side, with absence of Litten's sign and displacement of the heart toward the affected side, following diphtheria or aspiration of foreign bodies, or abdominal operations should suggest the diagnosis of massive collapse.

Differential Diagnosis.—*From Pulmonary Embolism.*—The onset is usually not so abrupt, hemoptysis is rare, diaphragmatic movement is less marked. The x-ray demonstrates the peculiar wedge-shaped shadow in most cases of pulmonary embolism. The heart is not displaced in pulmonary embolism.

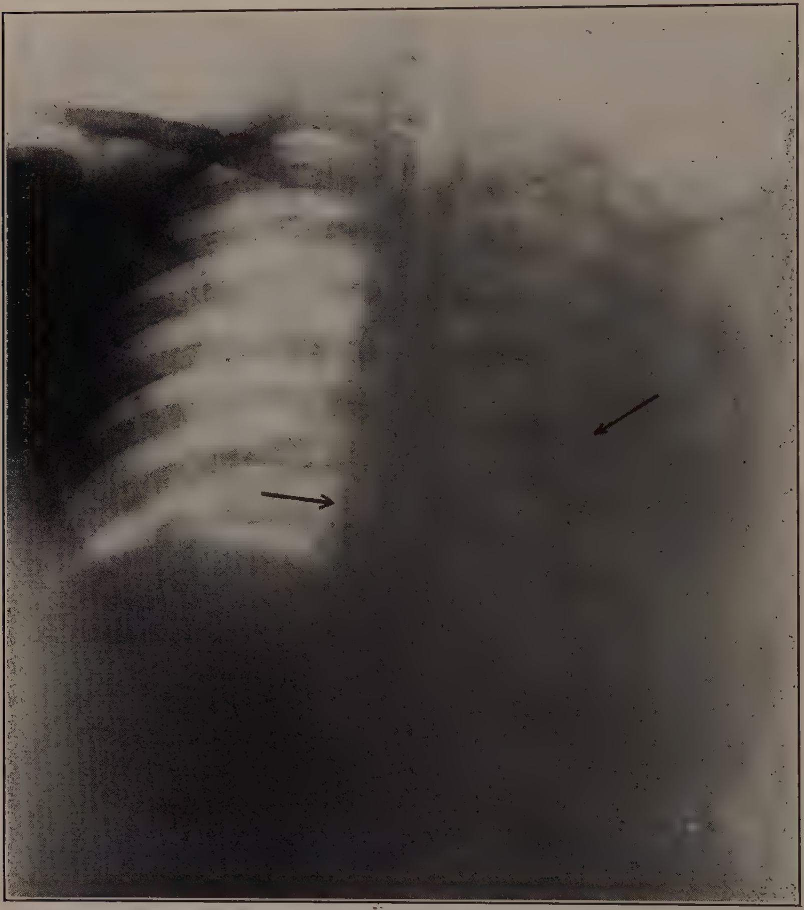

Fig. 291.—Radiograph of massive collapse of the lung. Arrow on right indicates absence of heart shadow. Arrow on left indicates area of collapsed lung.

Pneumothorax should be easily differentiated by the bulging of the intercostal spaces. Displacement of cardiac apex away from the affected area. Tympanitic percussion, note and coin sign.

EMPHYSEMA

Pulmonary emphysema represents a state of acute or chronic distention of the pulmonary alveoli with air, or an infiltration of the interalveolar tissues with air (interstitial emphysema). Clinically, five forms of pulmonary emphysema are recognized: *chronic hypertrophic emphysema, atrophic* or *senile emphysema, compensatory emphysema, acute vesicular emphysema,* and *interstitial* or *interlobular emphysema.*

CHRONIC HYPERTROPHIC EMPHYSEMA

Clinical Pathology.—Chronic hypertrophic emphysema, idiopathic or substantive emphysema, or the larged-lunged emphysema of Jenner, is a chronic disease, which is usually secondary to pathologic changes in the bronchi or lungs of considerable duration.

The precise pathogenesis of the disease has not been adequately explained, none of the theories heretofore elaborated proving convincing in all cases. Many cases of the disease appear to develop as a result of expiratory dyspnea of extensive duration, arising during the course of chronic bronchitis and bronchial asthma. Similarly, forced expiration with the glottis closed in players upon wind instruments and in glass blowers has been assumed to cause the disease, although this does not appear from statistics to have been a fertile source of the affection.

Occupations which entail heavy lifting, the patient inhaling deeply and then closing the glottis, seem to have some etiologic relation to hypertrophic emphysema. Likewise, occupations which necessitate the daily inhalation of irritating dusts are frequently attended by the disease.

It has been noted that there is a familial tendency to hypertrophic emphysema. In such families Cohnheim and Orth hold that there is a congenital hypoplasia of the elastic tissue elements of the lungs, predisposing to the development of emphysema.

Freund's theory of the pathogenesis of the disease holds that it is primarily a disease of the costal cartilages, which undergo an early hyperplasia and subsequent ossification, resulting in a rigid thoracic wall to which the emphysema is secondary. This theory is not generally accepted.

Hypertrophic emphysema is a bilateral disease, and involves both lungs to a similar degree. The distention of the lungs is general in all directions, but it attains its maximum degree in the anterior pulmonary borders, which, overlapping the heart, give rise to a diminution of the area of that organ which is in contact with the anterior chest and cause a consequent diminution in the area of cardiac dullness. The lungs are enlarged; they are pale; they are light and feathery to the touch; and they do not collapse readily when the thorax is opened at autopsy.

Microscopically the alveoli are observed to be abnormally large, and it is observed that in many instances the interalveolar septa are atrophic and have ruptured, leading to the production of larger cavities by the coalescence of several alveoli. With the destruction of the alveolar septa, the capillaries which they supported are destroyed, and the quantity of blood which is exposed to the air in the infundibula is commensurately diminished, resulting in deficient aeration of the blood, with consequent dyspnea and occasionally cyanosis upon moderate exertion.

The pleura covering the lung loses its pigmentation in patches, a condition which was termed by Virchow "albinism of the lung." The bronchi and bronchioles show signs of chronic bronchitis, and bronchiectatic dilatations are common.

The right heart gradually hypertrophies, as a result of the increased burden which is thrown upon it, and the tricuspid ring is usually enlarged so that the valve segments often fail to close the orifice completely.

Rupture of the atrophic walls of the infundibula subjacent to the pleura may result in the production of pneumothorax.

Physical Signs.—*Inspection.*—The facies of hypertrophic emphysema is fairly characteristic of the disease. The eyes are slightly prominent; the nose is somewhat thickened and cyanotic; while the head is thrown slightly backward in the effort to bring the accessory muscles of respiration into play. The neck is short and thick with prominent sternomastoids and trapezii.

Hypertrophic emphysema produces a definite alteration in the size and shape of the thorax, the barrel-chest of this disease. In this type of thorax, the anteroposterior diameter is increased to such a degree that it equals or exceeds the transverse diameter. The expansion of the chest is minimal, the thorax rising and falling vertically *en masse*. The expiratory movement is of longer duration than is the inspiratory effort.

The cardiac impulse is often invisible, and there is frequently visible epigastric pulsation, while pulsation of the jugular veins is common. In the late period of the disease, when cardiac failure is imminent, the patient is dyspneic, and frequently cyanotic. Not infrequently there is a delicate tracery of distended veins over the lower portion of the thorax, produced by intrathoracic obstruction to the venous return.

Litten's diaphragmtaic shadow is shortened in the established case of hypertrophic emphysema; the subject presents persistent chronic cough; and the terminal phalanges of the fingers are frequently clubbed.

Palpation.—Palpation reveals the deficient expansion of the thorax; and upon finger-tip palpation the intercostal spaces are hard and unyielding.

As a consequence of the excessive aerial content of the lungs, vocal fremitus is diminished in intensity over both sides of the thorax. However, in cases with marked chronic bronchitis, rhonchal fremitus is occasionally encountered. Also occasionally the intervention of the voluminous anterior pulmonary borders between the heart and the chest wall obscures the cardiac impulse.

Frequently there is a palpable impulse in the epigastrium which is due to the powerfully acting right ventricle. The liver is seldom displaced sufficiently to render its lower border freely palpable below the right costal arch; but in late cases, when right heart failure is imminent, palpation of the liver frequently reveals the systolic pulsation of tricuspid regurgitation. The spleen is rarely displaced in hypertrophic emphysema. In late cases palpation of the abdomen may reveal the presence of moderate ascites.

Palpation reveals the deficient expansion of the thorax; and upon finger-tip palpation the intercostal spaces are hard and unyielding.

Percussion.—Percussion yields a hyperresonant note over both lungs; and the limits of pulmonary resonance are extended in all directions, upward into the root of the neck, anteriorly, encroaching upon the area of cardiac dullness, and inferiorly over the areas of hepatic and splenic dullness. Percussion is useful in detecting downward displacement of the liver, and the presence of ascites by eliciting dullness in the flanks with tympany in the median line of the abdomen.

The respiratory excursion of the lungs, as determined by percussing the lower borders of the lungs during expiration and inspiration, respectively, is greately diminished in hypertrophic emphysema, rarely indeed exceeding half an inch.

Auscultation.—The respiratory sounds are distant, with prolongation of the expiratory phase. The expiratory murmur is faint and is not infrequently dotted with râles which are due to chronic bronchitis. The inspiratory murmur is always short, and often it is entirely inaudible during quiet breathing. Vocal resonance, like vocal fremitus, is impaired over both lungs.

The heart sounds as a whole are diminished in intensity, owing to the intervention of the distended anterior borders of the lungs between the heart and the chest wall. Of the individual sounds, the pulmonary sound is accentuated, as a result of the obstacle which is offered to the pulmonary circulation. Late in the disease the murmur of tricuspid regurgitation is often audible.

X-ray shows diminution of lung markings. The bronchial shadows often appear smaller and are not so clearly defined as in the normal lung.

Diagnosis.—The diagnosis of hypertrophic emphysema is frequently to be made during a casual examination. It rests upon the characteristic deformity of the thorax, associated with dyspnea and cyanosis, persistent chronic cough, the short or absent inspiratory murmur, and the prolonged, harsh expiratory murmur.

Differential Diagnosis.—*Chronic bronchitis,* with its chronic cough, shortness of breath upon exertion, and slightly prolonged expiratory murmur, simulates hypertrophic emphysema; but this disease is not attended in its early stages by deformity of the thorax, neither does it as a rule show general extension of the limits of pulmonary resonance, which forms so distinctive a feature of hypertrophic emphysema. However, the two diseases frequently coexist.

Pneumothorax, which in its incipiency may resemble hypertrophic emphysema, is a unilateral affection, which develops rapidly, affording a hollow and tympanitic note upon percussion, frequently also the succussion sound, the metallic tinkle, and the coin test, which serve to differentiate it from hypertrophic emphysema.

ATROPHIC EMPHYSEMA

Clinical Pathology.—Atrophic emphysema, a pulmonary disease in which the total bulk of the lung is decreased, is a senile change, a part of the general wasting of the tissues of the body incident to advanced age. The disease, if such it may be styled, is associated with persistent chronic cough, lasting over a period of many years, and associated with chronic shortness of breath upon exertion.

In the subject of atrophic emphysema the thorax is abnormally small, the obliquity of the ribs is increased, and the excursion of the thorax during respiration is distinctly limited.

The lung as a whole is reduced in size; the pleura is deeply pigmented; and the pulmonary parenchyma shows evidence of pulmonary congestion, edema, or infarction. The bronchial tubes frequently present dilatations, which are surrounded by areas of induration.

Microscopically there is atrophy and rupture of many of the interalveolar septa, leading to the formation of larger chambers by the coalescence of several smaller ones. The capillaries which the alveolar walls supported are destroyed in the areas of alveolar rupture, decreasing the quantity of blood which is exposed to the air in the infundibula.

Physical Signs.—*Inspection.*—In the subject of atrophic emphysema the thorax is small; the intercostal spaces are narrowed; the ribs pursue a more oblique course than normally; the supraclavicular and infraclavicular fossæ are of abnormal depth; and the thoracic excursion during respiration is slight, rendering Litten's shadow short or indistinct. The dyspnea of atrophic emphysema, instead of being chiefly expiratory as in hypertrophic emphysema, is mixed, the duration of the two phases of the respiratory cycle being approximately equal.

Palpation.—Upon palpation of the small thorax of the subject of atrophic emphysema vocal fremitus is as a rule moderately exaggerated, owing to the increased density of the lungs and to the diminution in their air content.

Percussion.—In atrophic emphysema the limits of pulmonary resonance are decreased in all directions. Even within the areas of resonance, as determined by percussion of the thorax, there is moderate impairment of the normal vesicular quality of the resonance, attributable over the apices to fibrosis and condensation of the pulmonary tissues, and over the bases posteriorly to edema, congestion, or infarction. The area of cardiac dullness is extended, as a result of shrinking of the lungs, exposing a large area of the heart to the anterior chest wall. Similarly, the upper limits of the areas of hepatic and splenic dullness occupy an abnormally high position, owing to contraction of the lungs.

Auscultation.—The respiratory murmur over the entire thorax is feeble with expiration prolonged; but occasionally bronchovesicular respiration is encountered. In the presence of chronic bronchitis, which often coexists with the emphysema, moist râles are in evidence over the bases of the lungs.

X-ray.—The bronchial markings are more pronounced and often show enlargements along their course. The lung structure is less dense except when there is marked fibrosis. The diaphragm is usually flattened.

Diagnosis.—The diagnosis of atrophic emphysema is readily made in an elderly subject with symmetrically diminished thorax, with a generally "dried up" appearance, chronic shortness of breath, and chronic cough of extensive duration.

COMPENSATORY EMPHYSEMA

Clinical Pathology.—In compensatory emphysema certain portions of a lung, or indeed an entire lung, contain an excess of air, as a result of a diminution of the air space of a portion of the same or of the opposite lung. Usually of transient duration, compensatory emphysema may become permanent. In inflammation of the terminal bronchioles with turgescence of the mucous membrane, obliteration of the lumen of the tubes is produced and the air in the infundibula is prevented from escaping during expiration, and the tendency is toward atrophy and ultimate rupture of the interalveolar septa. If the obstacle to the egress of the air persists, bullæ or air spaces of variable size are formed from the coalescence of several contiguous infundibula and a state of permanent emphysema is established.

A diffuse compensatory emphysema, involving an entire lung, usually results from massive pneumonia, chronic interstitial pneumonia, or a large pleural effusion interfering with the expansion of the opposite lung during inspiration. Localized areas of compensatory emphysema occur in a lung which is the seat of atelectasis, the multiple patches of consolidation of bronchopneumonia and phthisis, and in the presence of local fibrosis of the lung.

Physical Signs.—The physical signs of compensatory emphysema vary with the distribution of the emphysema and its degree, whether involving an entire lung or merely portions of a lung; and in the latter event, varying with the extent of the lung which is rendered emphysematous.

Inspection.—Upon inspection in a case in which the entire lung is compensatorily emphysematous, there is unilateral bulging of the thorax corresponding to the side of the emphysema; and this bulging is accentuated by the retraction of the opposite side, which is diminished as a result of disease of the lung which has occasioned the emphysematous state of its fellow. Small areas of localized compensatory emphysema produce no alteration in the contour of the thorax.

Palpation.—Vocal fremitus over a unilateral compensatory emphysema is diminished in intensity as a result of the rarefaction of the pulmonary tissues. Over the opposite lung the fremitus is of variable intensity, being influenced by the state of the lung which has resulted in the compensatory emphysema of its fellow. In the presence of consolidation the fremitus is commonly exaggerated, whereas in the presence of extensive pleural effusion it is totally abolished over the effusion.

Percussion.—Percussion in the case of compensatory emphysema of an entire lung yields a hyperresonant note over the emphysema, with dulling of the note over the crippled lung. Localized emphysema of limited extent produces no alteration in the normal resonance of the lung upon percussion.

Auscultation.—Over an emphysematous lung the respiratory murmur is puerile, with slight prolongation of the expiratory phase. Over the opposite lung the murmur is influenced by the condition which was responsible for the unilateral compensatory emphysema, being bronchovesicular or frankly bronchial in the presence of consolidation of the lung, and being absent over the distribution of a large pleural effusion.

X-ray shows little of diagnostic value. The lung structure is less dense, the diaphragm is well outlined, and on fluoroscopic examination is seen to have a somewhat increased excursion.

Diagnosis.—A diagnosis of compensatory emphysema can rarely be made upon the physical signs alone, as these are frequently atypical. The history of the case should be elicited, with the discovery of some adequate cause of compensatory emphysema. Hyperresonance of one side of the thorax with puerile respiration and vicarious expansion, with dullness of the opposite side with possibly retraction of the thorax, and with deficient expansion speaks strongly in favor of compensatory emphysema.

ACUTE VESICULAR EMPHYSEMA

Clinical Pathology.—Acute vesicular emphysema is a condition in which the infundibula are acutely distended from expiratory efforts or fits of coughing in the presence of an obstacle to the free egress of air from the lungs. The disease is apt to develop during bronchopneumonia, bronchial asthma, tracheal and bronchial stenosis, and during the extreme dyspneic attacks of cardiac failure.

The distention of the lungs occurs abruptly; but the alveoli are merely transiently distended; and large cavities are not produced by rupture of the interalveolar septa as in hypertrophic emphysema, for recovery ensues or death occurs ere this change supervenes.

Physical Signs.—The physical signs of acute vesicular emphysema resemble in the main those of the hypertrophic form of the disease. There is general extension of the areas of pulmonary resonance; the percussion note is hyperresonant or even tympanitic; and upon auscultation there are sibilant râles universally over both lungs, with prolongation of the expiratory murmur. However, these signs are transient, and auscultation of the heart reveals no accentuation of the pulmonic second sound.

X-ray is of little diagnostic value.

INTERSTITIAL EMPHYSEMA

Clinical Pathology.—In interstitial emphysema a variable quantity of air or gas is present in the interlobar or interlobular septa, or beneath the pleura. Air may gain access to these regions as a result of traumatism or as a consequence of violent expiratory efforts, when rupture of the lining epithelium affords ingress of air to the subjacent structures. It has also occurred during convulsions, during parturition, and as a sequence of straining at stool. It may be caused by ulceration of the bronchi, or by abscess or gangrene of the lung. Interstitial emphysema in the newly born has been caused during violent efforts to mechanically inflate the lungs, and it has resulted from spasmodic closure of the glottis.

When air or gas has gained access to the stroma of the lung, it collects in the form of beads or bullæ of variable size. In certain instances they are very small, while in other cases they attain to the size of a walnut. The beads of air are prone to make their way toward the root of the lung and into the mediastinum and thence to pass upward along the trachea and to appear beneath the skin of the base of the neck. Or the opposite sequence of events may be observed. Following tracheotomy wounds, air has entered the tissues and burrowed downward into the roots of the lungs and invaded the stroma of these organs. Not infrequently the beads of air form bullæ immediately subjacent to the pleura, and by rupture produce limited pneumothorax.

Physical Signs.—Interstitial emphysema produces few physical signs, and the condition may escape detection entirely during a casual examination. When the air makes its way upward into the root of the neck and appears subcutaneously, it is apt to produce a protrusion, which, upon palpation with the finger-tips, yields a peculiar crackling crepitus. Large beads of air subjacent to the pleura are apt to yield a sound closely simulating the pleural friction sound.

X-ray shows an area of lessened density surrounded by a narrow margin of increased density somewhat resembling a cavity but with well-defined lung markings showing through.

ABSCESS OF THE LUNG

Clinical Pathology.—The cases of pulmonary abscess which come before the clinician may be classified as bronchogenic, pneumogenic, or extraneous in origin.

The cases of bronchogenic origin develop as the consequence of the aspiration into the bronchial tubes of infectious material during or subsequent to operative procedures upon the buccal or nasopharyngeal structures, and as the result of the lodgment in the bronchi of aspirated foreign bodies.

The initial alteration in the postoperative case is the establishment of a local bronchitis at the site of implantation of the aspirated infectious material. Local ulceration of the bronchial mucous membrane ensues, and is attended by thinning and yielding of the bronchial wall, with the consequent production of bronchiectasis. With the further disintegration of the bronchial wall and by the extension into the bronchioles and infundibula of the septic bronchitis, a deglutition or aspiration bronchopneumonia is established, which is prone to eventuate in pulmonary abscess.

When an aspirated foreign body becomes arrested in a bronchus, local inflammation and ulceration of the bronchial mucous membrane occurs at the site of lodgment, with the development, as demonstrated by Cohn, of bronchiectasis at this point, and not distal to the occlusion as was formerly believed. The progressive disintegration of the bronchial wall in these cases, combined with the implantation of pyogenic organisms, not infrequently is productive of pulmonary abscess.

In the pneumogenic group of cases are comprised the cases of pulmonary abscess arising as a sequence of lobar pneumonia, bronchopneumonia complicating influenza or other acute infectious diseases, pulmonary tuberculosis, syphilis, or actinomycosis of the lung.

Pulmonary abscess complicating lobar pneumonia occurs in two forms; namely, as a widespread purulent infiltration of the lung, and as single or multiple circumscribed abscesses. A purulent infiltration of the lung represents an extreme grade of gray hepatization with tardy resolution. Tuffier found that among forty-nine cases of pulmonary abscess treated surgically, in twenty-three lobar pneumonia only could be assigned as the cause; while, in a series of one hundred cases, Wessler found thirty-seven cases to be distinctly of pneumonic origin.

Chronic ulcerative phthisis in the course of its evolution occasionally results in pulmonary abscess. The tuberculous abscess is discrete and circumscribed, develops during the latter part of the course of the disease, and is associated with caseation and cavity formation.

The cases of pulmonary abscess of extraneous origin comprise the cases of pyemic origin, and the ''perforating abscesses'' of Stokes. When an infectious embolus from an area of osteomyelitis or from a vegetation upon one of the cusps of a cardiac valve which is the seat of malignant endocarditis occludes a branch of the pulmonary artery, an infarct of the pulmonary tissues is apt to form and through secondary infection to eventuate in abscess of the lung. Pyemia is usually attended by the formation of multiple pulmonary abscesses of limited dimensions. They are usually situated in the periphery of the lung, immediately subjacent to the visceral pleura; and they assume primarily a conical form, with the base of the cone directed toward the pleura.

Stokes designated as ''perforating abscesses'' of the lung the numerous cases of pulmonary abscess in which collections of pus from extraneous sources involve the lung secondarily by contiguity of structure. The source of the pus in this class of cases is variable. It may originate in an abscess of the thoracic wall, an abscess of the liver, a perforating ulcer of the stomach, carcinoma of the esophagus, or suppuration of the mediastinal glands.

Perforating wounds of the thorax occasionally eventuate in abscess of the lung. A penetrating missile may thus carry into the lung fragments of wearing apparel or other bacteria-laden material, which results in suppuration of the pulmonary parenchyma.

In its dimensions a pulmonary abscess may be quite small or it may be very extensive, indeed, may involve the major part of a lobe of the lung. The pulmonary abscess is usually solitary; but in pyemic cases multiple abscesses of limited dimensions are commonly dispersed widely throughout the lungs.

The shape of the pulmonary abscess is very irregular; and an abscess is occasionally divided by tissue bands into two or more chambers or loculi. The internal surface of the abscess is irregular, presenting mural shreds and occasionally excrescences protruding into the cavity from a pyogenic membrane, which in turn is supported by a more or less well-defined fibrous capsule, which in the latter stages of the disease becomes the point of inception of a variable degree of insular sclerosis of the involved lobe of the lung.

Pulmonary abscess is usually, though not invariably, situated in the peripheral portion of the lung, subjacent to the visceral pleura; and in the majority of cases it occupies the lower lobe of the lung. In the case of the postoperative abscess of bronchogenic origin, however, the abscess is not infrequently situated in the upper or middle lobe, especially in the case of children. Wessler found, upon analysis of the distribution of the lesions in his series, that among the postoperative aspiration cases the abscess was localized in the upper lobe in eighteen cases, in the lower lobe in nine cases, and in the middle lobe in one instance; while among the nonaspiration cases the abscess was situated in the upper lobe in twenty-four cases, in the lower lobe in forty-four cases, and in the middle lobe in three of the subjects.

The contents of a pulmonary abscess is purulent, containing shreds of elastic tissue, bacteria, cholesterin crystals, and necrotic débris. The organisms which are commonly present comprise the pyogenic streptococci and staphylococci, the pneumococcus, bacillus influenzæ, and the bacillus of Friedländer, with the occasional association of the bacillus pyocyaneus, bacillus coli communis, or the bacillus tuberculosis.

Spontaneous evacuation of a pulmonary abscess is apt to occur by rupture into an adjacent bronchus, in which event the purulent contents of the abscess cavity is partially expectorated, the shreds of elastic tissue in the sputum aiding materially in the diagnosis; or, if the abscess is situated immediately subjacent to the pleura, this membrane may be penetrated, permitting the abscess to discharge into the pleural cavity with the consequent induction of pyopneumothorax.

When an intact pulmonary abscess occupies the peripheral portion of the lung, closely subjacent to the visceral pleura, pleuritis is a constant accompaniment of the disease. The surface of the inflamed pleura is early clothed with fibrinous or fibrinopurulent exudate, occasionally with the subsequent supervention of frank empyema.

Bronchiectasis and pulmonary abscess are very frequently concomitant diseases. The existence of uncomplicated bronchiectasis lends a strong predisposition to the development of pulmonary abscess; while pulmonary abscess of bronchogenic origin induces a variable degree of bronchiectasis during its evolution.

Postpneumonic pulmonary abscess, whether it assumes the form of purulent infiltration or of multiple circumscribed abscess cavities, produces chronic induration of the lung with diminution in its total bulk, marked destruction of capillary circulation, and multiple bronchiectases.

Septic thrombosis of the cerebral vessels, with the induction of abscess of the brain, has proved a fatal complication in a considerable number of cases of pulmonary abscess.

Physical Signs.—The physical manifestations of pulmonary abscess vary with the character and with the distribution of the lesion. Multiple small abscesses, distributed widely throughout the lung, and likewise a very diffuse purulent infiltration, yield no distinctive physical signs by which alone the condition could surely be detected. Similarly, a solitary circumscribed abscess, which is situated in the depths of the lung and which is separated from the thoracic wall by a tolerably thick bed of crepitant pulmonary tissue, successfully eludes detection during a physical examination. It is in the cases in which the abscess is situated in the periphery of the lung, or when it is superimposed upon one of the larger bronchial tubes that a diagnosis is most readily achieved by the methods of physical examination.

Inspection.—The subject of pulmonary abscess generally presents an appearance of septic intoxication, with pallor of the skin and mucous membranes, which is not infrequently associated with a variable degree of cyanosis of the lips, ears, and finger tips. If the abscess is at all extensive, there is acceleration of the respiration, which is shallow, and there is visible impairment of expansion of the side of the thorax corresponding to the disease of the lung.

The decubitus of the patient will vary with the site of the disease. When the abscess is situated in the lower lobe, the patient assumes the upright posture in order to maintain the sensitive mucous membrane of the communicating bronchial tubes free from the purulent contents of the abscess cavity. When, on the contrary, a solitary abscess involves the upper lobe, the subject is prone for similar reasons, to assume the recumbent or semirecumbent posture in bed.

When contact of the contents of the cavity with the bronchial mucous membrane occasions a paroxysm of cough, or when a closed abscess cavity ruptures into a bronchus, copious expectoration of fairly characteristic sputum ensues. The sputum occasionally possesses a somewhat sweetish odor, though the rule is rather for foul expectoration, which is raised in mouthfuls; but the sputum of pulmonary abscess never attains the excessive foulness of the expectoration attending pulmonary gangrene. Frequently the sputum contains shreds of elastic tissue; and, in the cases of extensive duration, crystals of cholesterin may be found.

Clubbing of the fingers is not uncommon in abscess of the lung, and in this acute disease these changes become manifest much earlier in the course of the disease than in phthisis.

Palpation.—When pulmonary abscess is situated in the peripheral portion of the lung, closely subjacent to the visceral pleura, friction fremitus which is produced by a coincident pleuritis may occasionally be detected upon palpation of the thorax. Finger-tip palpation of the intercostal spaces over this area yields a sensation of increased resistance when the abscess cavity is filled, which gives place to a sensation of markedly diminished resistance upon partial or complete evacuation of the purulent contents of the subjacent cavity.

Numerous transient variations in the intensity of vocal fremitus are encountered upon palpation of the thoracic surface over an extensive pulmonary abscess which occupies the periphery of the lung. In the cases in which the abscess cavity is closed, there is persistent diminution or abolition of the vocal vibrations in the localized area of increased resistance. When, however, the abscess possesses a patent bronchial communication by which its purulent contents is evacuated at intervals, the variations in vocal fremitus are striking. Under these circumstances tactile fremitus is absent over the filled cavity, and reappears in exaggerated form upon partial evacuation of the secretions of the abscess cavity.

Percussion.—Pulmonary abscess involving the peripheral portion of the lung yields dullness upon percussion, when it is filled, with hyperresonance over the adjacent areas as a result of compensatory emphysema of the neighboring air vesicles. When, however, the abscess has recently discharged a considerable portion of its purulent contents, the sound elicited by forcible percussion of the thorax is tympanitic or amphoric. In other words, the sounds elicited now are those of pulmonary excavation; and, if the abscess cavity possesses a patent bronchial communication, the examiner may be successful in eliciting a cracked-pot sound or the phenomena of Wintrich, Friedreich, or Gerhardt.

Auscultation.—In the presence of abscess of the lung, whether the cavity be filled or empty, auscultation of the zone surrounding the disease reveals the presence of numerous crepitant and subcrepitant râles, which arise as a consequence of a local bronchiolar inflammation and pulmonary edema. If the abscess is of considerable dimensions and is filled with secretions, the vesicular murmur is abolished over the corresponding area of the thoracic surface, while vocal resonance is markedly impaired in the same area. If, however, the abscess cavity be only partially filled, blowing bronchial or amphoric breathing is encountered, together with exaggerated vocal resonance upon auscultation.

X-ray.—Pulmonary abscess yields, in the majority of cases, a very definite shadow, which is of service in accurately localizing the disease. When the abscess cavity is partially empty a fluid level is visible which changes when the radiographs are taken in a different position. This is especially well brought out by the instillation of iodized oil. In any equivocal case the examiner should be guided by the physical findings rather than by the radiogram.

Diagnosis.—Signs of sepsis such as chills, fever, and colliquative sweats, with copious expectoration of purulent sputum, combined with signs of pulmonary excavation, usually involving a lower lobe of the lung, following upon one of the conditions which may be provocative of pulmonary abscess, suggest the diagnosis of abscess of the lung. When a patient with lobar pneumonia continues to run a temperature with sweats after the crisis of the disease has occurred, one should think of the possible supervention of a purulent infiltration upon the pneumonic state.

Thoracic pain is frequently a prominent symptom in the cases in which the abscess is subpleural with complicating inflammation of this membrane. In cases of pyemic origin signs referable to the small, disseminated abscesses are often obscured by the general symptoms of pyemia. In the later stages of the disease emaciation is striking, with pallor of the skin and cyanosis of the lips and digits, while the patient is frequently somnolent or delirious. When, in closed cases, a bronchus is perforated and copious expectoration of purulent sputum containing elastic fibers occurs, the diagnosis is assured. Upon perforation of the visceral pleura by a subpleural abscess, signs of pyopneumothorax are in evidence.

Differential Diagnosis.—Pulmonary abscess must be differentiated from *bronchiectasis, empyema, pulmonary gangrene,* and *chronic ulcerative phthisis.*

Bronchiectasis is attended by copious expectoration of mucopurulent or purulent sputum, with frequently signs of pulmonary excavation. The sputum of bronchiectasis, however, is free from elastic shreds; and the history and course in the two affections are entirely dissimilar, bronchiectasis being a distinctly chronic disease of extensive duration and mild symptomatology, while pulmonary abscess is an acute disease of relatively brief duration and stormy course.

Empyema of extensive duration produces signs of septic intoxication with pallor, emaciation, chills, and sweats; but in empyema the disproportion between the two sides of the thorax is more extreme; there is demonstrable an extensive area of flatness laterally and posteriorly, with absence of respiratory sounds; and the sputum does not contain elastic fibers.

X-ray will localize this area either in the lung or pleural cavity.

From *pulmonary gangrene,* which in its evolution also produces cavitation, abscess is differentiated by the absence of the extreme fetor of the expectoration of the former disease, a fetor which rapidly pervades a house or hospital ward and immediately suggests the diagnosis of gangrene. Moreover, the sputum in gangrene of the lung contains shreds of decomposing pulmonary tissue, which frequently present the characteristic structure of the alveoli of the lung.

In *chronic ulcerative phthisis* the vomicæ are prone to appear in the upper lobes, where they are encompassed by zones of infiltrated and consolidated pulmonary tissue. Phthisis is a chronic disease, and bilateral disease commonly can be detected in the presence of cavitation. In every case repeated examinations of the sputum should be made for the detection of the tubercle bacillus.

GANGRENE OF THE LUNG

Clinical Pathology.—As invasion of the lung in which the circulation is normal by pyogenic bacteria or infectious material from the upper respiratory passages is apt to eventuate in abscess of the lung, so a similar invasion of the lung in which the circulation is deficient is prone to result in pulmonary gangrene. It is upon this impairment of the vascular integrity of the pulmonary tissues that rests the predisposition to pulmonary gangrene in subjects of diabetès mellitus, chronic valvular disease of the heart, and chronic alcoholism.

Gangrene of the lung, as first recognized by Laënnec, occurs in two forms; namely, as circumscribed pulmonary gangrene, and as diffuse gangrene of the lung. The two classes of cases differ as widely in their mode of inception and evolution as they do in their physical manifestations.

Circumscribed pulmonary gangrene has its point of inception in the pulmonary arteries, in the bronchi, or in cavities of bronchiectatic or tuberculous origin.

Embolic gangrene of the lung, in which infectious embolism arises as a result of malignant endocarditis, osteomyelitis, or suppurative otitis media, is of arterial origin. In this group of cases the area of gangrene is situated peripherally, not infrequently closely subjacent to the visceral pleura. Occasionally pulmonary gangrene ensues upon noninfectious embolism of the pulmonary artery.

Pulmonary gangrene of bronchial origin follows the lodgment of aspirated foreign bodies in the smaller bronchial tubes, or it ensues upon the aspiration into the finer bronchioles of infective material from the upper respiratory passages. Cohn has demonstrated that following the lodgment of a foreign body in the bronchial system, inflammation and ulceration of the mucous membrane with subsequent disintegration of the bronchial wall occurs, with the establishment of bronchiectasis, pulmonary abscess, or pulmonary gangrene as the concomitant factors may determine. Similarly, the aspiration of infective material from the upper respiratory passages is prone to induce an aspiration pneumonia or a putrid bronchitis at the area of arrestment, in which, in the words of Hensel, "the putrid properties of the aspirated particles lend a gangrenous tendency to the catarrhal pneumonia which they produce."

Gangrene of the lung arising from pulmonary excavation is encountered as a sequence of bronchiectasis with the accumulation of purulent secretions, chronic ulcerative phthisis with cavity formation, and abscess of the lung.

Traumatism in the form of penetrating wounds of the lung has caused pulmonary gangrene, as has also trauma in the form of contusion or laceration of the pulmonary tissue without perforation of the chest wall.

Diffuse gangrene of the lung is an occasional terminal complication of lobar pneumonia; but more frequently it arises as a sequence of catarrhal pneumonia of the aspiration or deglutition type. The disease has similarly been induced by carcinoma of the lung, and it has arisen as a result of pulmonary compression by aortic aneurysm and mediastinal tumor.

In the circumscribed form of pulmonary gangrene the disease assumes either the form of a single area of pulmonary mortification, or of multiple areas of gangrene, separated by partially crepitant pulmonary tissue. The disease most frequently involves the inferior lobe, in which it is prone to develop in the periphery of the lung. In the early stage of the disease the gangrenous area is reddish-brown, green, or black, and is firm; but in the further evolution of the morbid process softening and destruction of the pulmonary tissues occur, with the production of a central excavation with irregular, rugged walls, and which contains a greenish offensive fluid. In the subpleural cases the finger of the pathologist penetrates readily into the area of gangrenous tissue. In the cases in which the gangrene is more centrally situated, the gangrenous area is surrounded by a zone of pulmonary tissue which is the seat of local pulmonary congestion with imperfect consolidation or splenization, while adjacent to the area of congestion the lung is the seat of collateral edema.

In rapidly spreading gangrene of the lung a branch of the pulmonary artery is apt to be eroded, eventuating in pulmonary hemorrhage, which may be so copious as to prove fatal. Owing to the usual peripheral distribution of the pulmonary lesion, the visceral pleura is also apt to be perforated, with the establishment of pyopneumothorax.

Bronchitis is a constant concomitant state, as a result of the continuous irritation of the bronchial mucous membrane by the fetid contents of the gangrenous cavity. Gangrene of the lung is likewise occasionally associated with abscess of the brain. In pulmonary gangrene of bronchial origin varying degrees of bronchiectasis are frequently present in the lung, and pulmonary abscess is not infrequently complicated by gangrene of the adjacent portions of the lung.

In pulmonary gangrene there is a rapid reduction in the erythrocytes and a commensurate reduction in the hemoglobin of the circulating blood stream; and, in cases of pyemic origin, septicemia is not infrequently demonstrable. Leucocytosis is the exception and not the rule. The course of the disease is brief and stormy, the subject succumbing in the vast majority of cases in a period varying from a few weeks to a few months from the onset of his disease.

Physical Signs.—The physical signs of pulmonary gangrene vary with the site and the distribution of the lesions which are present in the lung. When the gangrenous area occupies the peripheral portion of the lung, and when there is active pulmonary excavation, the physical signs are those of cavity, the manifestations of the disease varying naturally as the cavity possesses a patent bronchial communication or is closed. When, on the contrary, the area of gangrene is centrally situated, is devoid of all communication with the bronchial system, and is separated from the thoracic parietes by a tolerably thick bed of crepitant pulmonary tissue, the physical signs are anomalous or entirely wanting. In the majority of cases, in addition to the signs which are attributable to the gangrenous process, the characteristic signs of coexistent bronchial inflammation and partial consolidation are in evidence.

Inspection.—The subject of pulmonary gangrene commonly presents a picture of extreme septic intoxication. Emaciation is rapid and the skin is pallid, frequently with cyanosis of the lips, auricles, and digits. The patient is wracked by persistent paroxysms of cough, arising as a result of irritation of the sensitive bronchial mucous membrane by the acrid contents of the gangrenous cavity.

The patient instinctively assumes the posture in which the contents of the cavity are prevented from coming in contact with the mucosa of the communicating bronchi. When the gangrene attacks an inferior lobe of the lung, the patient is prone to assume the semirecumbent posture; whereas, when the area of gangrene is situated in an upper lobe, the subject rests most comfortably in the recumbent posture with the shoulders lower than the remainder of the trunk.

Hemoptysis is frequently to be noted in gangrene of the lung. It may consist in an admixture of traces of blood with the sputum, or it may occur in the form of copious pulmonary hemorrhages.

The breath is exceedingly foul in pulmonary gangrene, a fact which constitutes a valuable sign of the disease when considered in conjunction with the sputum, which is characteristic of the condition. The latter is abundant; and, upon standing for several hours, separates into three strata: an inferior stratum composed of heavy or brown sediment containing shreds of elastic tissue, bacteria, blood cells, fatty-acid crystals, leucin and tyrosin; a median layer composed of brown or greenish serous fluid; and a superior stratum of grayish-brown froth.

Palpation.—In the subject of pulmonary gangrene the inspiratory excursion of the thoracic wall is strikingly diminished; the respiratory movements are at the same time more frequent and often irregular or jerking; and the lifting power of the thoracic cage is practically nil.

When, in the presence of central gangrene of the lung, the adjacent zone of infiltration and consolidation attains to the visceral pleura, there is frequently appreciable to the palpating hand a minor exaggeration of vocal fremitus, an increase which is further exaggerated when adhesions have been established between the visceral and the costal pleura. When pulmonary gangrene originates in the cavities of chronic ulcerative phthisis, bronchiectasis, or pulmonary abscess, occupying the peripheral portion of the lung as these lesions are prone to do, tactile fremitus is markedly exaggerated over the site of the lesion, and to a lesser degree over the adjacent zone of consolidated pulmonary tissue.

Percussion.—The percussion findings in cases of pulmonary gangrene have been variable; and, unless they are construed in connection with other manifestations of the disease, they are apt to result in confusion, especially in cases of closed or latent gangrene of the lung. When the gangrenous area is covered by a bed of densely consolidated pulmonary tissue, the examiner encounters dullness upon percussion, a dull note upon which there is not infrequently engrafted a tympanitic quality when the gangrene has resulted in pulmonary excavation. A suggestive feature of the dullness which attends pulmonary gangrene resides in the fact that the area of impaired resonance frequently extends uninterruptedly over an interlobar fissure, as gangrene is frequently not limited to a single pulmonary lobe, but is prone to involve contiguous portions of adjacent lobes of the lung.

Careful percussion of the thoracic surface will frequently reveal a zone of moderate hyperresonance surrounding the area of dullness which is induced by the pulmonary consolidation, a hyperresonance which is to be attributed to local compensatory emphysema of the pulmonary alveoli in these regions.

Auscultation.—The vesicular murmur in pulmonary gangrene is frequently distant and feeble over the entire thorax as a result of the general debility of the subject of the disease. Occasionally bronchial breathing is to be elicited over an area of consolidation or pulmonary excavation. The crepitant râle is very frequently in evidence as an indication of the concomitant bronchial inflammation.

In the presence of gangrene of the peripheral portion of the lung a fine pleural friction sound is occasionally to be detected, a rub which develops as a consequence of a complicating local pleuritis.

In cases of embolic origin auscultation of the precordia not infrequently reveals the murmurs of coexistent valvular lesions.

X-ray serves to determine the size and shape of the area involved. This appears as an area of density, often lighter in the center, and fading into normal lung structure at the periphery.

Diagnosis.—The signs and symptoms upon which the diagnosis of pulmonary gangrene must be based comprise progressive emaciation and prostration, dyspnea, elevation of temperature, rapid cardiac action, foulness of the breath, the suggestive decubitus of the patient, and the characteristic sputum, when it is present. The occurrence of hemoptysis and the detection of shreds of disintegrated alveolar tissue in the expectoration are determining factors in arriving at a diagnosis. But in cases of latent pulmonary gangrene the breath is not foul, neither is the sputum characteristic, as in this class of cases the area of gangrene is definitely circumscribed and is devoid of any communication with the bronchial system. This type of the disease is prone to develop in diabetic and insane patients, and in these cases an error in diagnosis is almost certain to arise. A history of diabetes, bronchopneumonia, valvular heart disease, or bronchiectasis should serve in some measure to place the physician upon his guard when interpreting obscure physical findings in a debilitated subject.

Differential Diagnosis.—Mere foulness of the breath in any suspected case of pulmonary gangrene does not suffice to establish a positive diagnosis, as a foul breath may result from putrid bronchitis, decomposition of the purulent contents of bronchiectatic cavities, or even from carious teeth, in the absence of gangrene of the lung.

In *pulmonary abscess,* while the sputum is abundant and purulent, the odor of the breath is sweetish rather than fetid, as in gangrene. Moreover, the sputum in abscess is copious, occurring frequently as "mouthful expectoration;" it more frequently contains bacteria and cholesterin crystals; and shreds of alveolar tissue are not as abundant as in the case of gangrene.

Chronic ulcerative phthisis with cavity formation is attended by foulness of the breath and purulent expectoration containing elastic fibers; but the sputum in this disease contains tubercle bacilli, and the foulness of the breath never approaches the stench of pulmonary gangrene. The cavities of phthisis are prone to involve the upper lobe, and at this advanced stage of the disease bilateral involvement of the lungs is demonstrable. Unfortunately, in gangrene of the lung acid-fast bacilli closely resembling the tubercle bacillus morphologically are apt to be encountered in the sputum and to result in an erroneous diagnosis.

In *putrid bronchitis* the breath is excessively foul; but in this disease the expectoration which is raised is free from elastic shreds, while it contains characteristic elements in the form of Dittrich's plugs. While the prostration attending certain cases of putrid bronchitis is remarkable, it does not equal that of advanced gangrene of the lung.

TUMORS OF THE LUNG

Clinical Pathology.—Tumors of the lung and its investing pleura comprise sarcoma, carcinoma, and endothelioma. They may arise primarily in this organ, or may be secondary to tumor of adjacent structures or remote organs of the body.

Primary sarcoma of the lung is quite rare, the majority of pulmonary sarcomata representing metastases from neighboring organs. However, primary sarcoma is occasionally noted, arising from the pulmonary connective tissue or bronchial lymph nodes. The growth usually assumes the spindle-cell or round-cell variety of sarcoma. The neoplasm may assume the form of a single tumor occupying the root of the lung, or several round or oval growths may be distributed throughout the lung. Metastasis usually occurs to the mediastinal glands.

Primary carcinoma of the lung is likewise rare, only 125 cases being reported by Wiczkowski's analysis of 58,497 autopsies. The growth may spring from the bronchial epithelium, bronchial mucous glands, or the alveolar epithelium. Primary carcinoma may assume the form of a single, discrete tumor in the hilus region, or it may be represented by a diffuse carcinomatous infiltration of the lung. It has been stated that primary carcinoma has a predilection for the right lung, but Adler and Weller observed an almost equal involvement of the two lungs. Metastasis is frequently widespread, but it was absent in 33 of 374 cases studied in this respect by Adler.

Endothelioma usually springs from the pleura, arising from the cells of the subpleural lymph spaces. The growth extends over an extensive area of the membrane, having its inception as multiple small nodules, which subsequently fuse, and invade the subjacent pulmonary tissues. Metastasis to the regional lymph nodes is frequent, and the pericardium or peritoneum may become secondarily involved.

Secondary tumors of the lung include the three forms enumerated, and in addition in rare instances hypernephroma and choriocarcinoma or malignant deciduoma invade the lungs secondarily.

Males are the subjects of pulmonary malignancies more frequently than are female subjects, sarcoma predominating in young subjects, and carcinoma affecting adults with greater frequency.

Pulmonary malignant tumors are very frequently attended by effusion into the pleural cavity, which is apt to assume a hemorrhagic character, and which may for a time obscure the true nature of the malady.

The onset of the disease is insidious in the majority of cases, with a gradual decline in the strength and weight of the patient. There are isolated cases, however, in which the onset is acute, and the course is of brief duration.

Dyspnea is an early symptom in many cases, varying in intensity from moderate embarrassment of the respiratory function to extreme distress attended by cyanosis. The dyspnea is commonly attended by cough, which is particularly marked when the bronchi and trachea are compressed by the growth. Expectoration is the rule, the sputum possessing no characteristic features. It may be purely mucoid, mucopurulent, purulent, or hemorrhagic, and Stokes has described a very gelatinous red *currant-jelly* sputum, which was assumed to be pathognomonic of pulmonary neoplasm. Occasionally, when the tumor is undergoing disintegration, shreds of tissue may be found in the expectoration.

Pain is almost a constant feature of the disease, assuming the form of a stitch in the side, or of dull boring pain simulating the pain of aneurysm. In other instances pain is elicited only upon pressure exerted in the intercostal spaces.

Hoarseness and dysphagia are occasionally noted as a result of compression of the recurrent laryngeal nerve or of the trachea.

The course and duration of the disease vary with the type and with the rate of development of the neoplasm. The minimum duration noted has been three weeks, and the maximum four years.

Physical Signs.—The physical signs of tumor of the lung may be caused by the presence of the tumor or they may be due to the accompanying pleural effusion, when the latter is present. In the latter event the signs of pleurisy with effusion overshadow the other signs which might be present.

The superficial veins of the thorax and the veins of the neck may be tortuous and distended, owing to compression of the superior vena cava within the thoracic cavity. The contour of the thorax is altered. In the case of a very large growth there is unilateral bulging and widening of the intercostal spaces; whereas in the case of a small growth, causing collapse of the adjacent pulmonary tissues, or owing to traction by adhesions, there is restriction of the expansion of the thorax and local depression of the chest wall.

Vocal fremitus is occasionally exaggerated, while at other times it is diminished in intensity. A hyperresonant note is elicited upon percussion when the tumor has broken down and resulted in pulmonary excavation; whereas dullness or flatness is elicited over a large growth which involves an extensive area of the lung.

The breath sounds are suppressed in many instances; but in the presence of a growth of some size which is superimposed upon a large bronchus, the breath sounds are bronchial. In the presence of pulmonary excavation the breathing is apt to be amphoric.

X-ray may reveal a dense area with sharply defined borders.

Diagnosis.—In primary cases a diagnosis is made with difficulty; but the presence of strictly unilateral signs, attended by glandular enlargement, is suggestive of pulmonary neoplasm. The x-ray is of material aid in the diagnosis. In the case of carcinoma of the lung occasionally carcinomatous tissue is demonstrable in the sputum; and late in the course of the disease the growth is apt to perforate the chest wall. In a suspected case of pulmonary neoplasm, mediastinal tumor and aortic aneurysm must be eliminated.

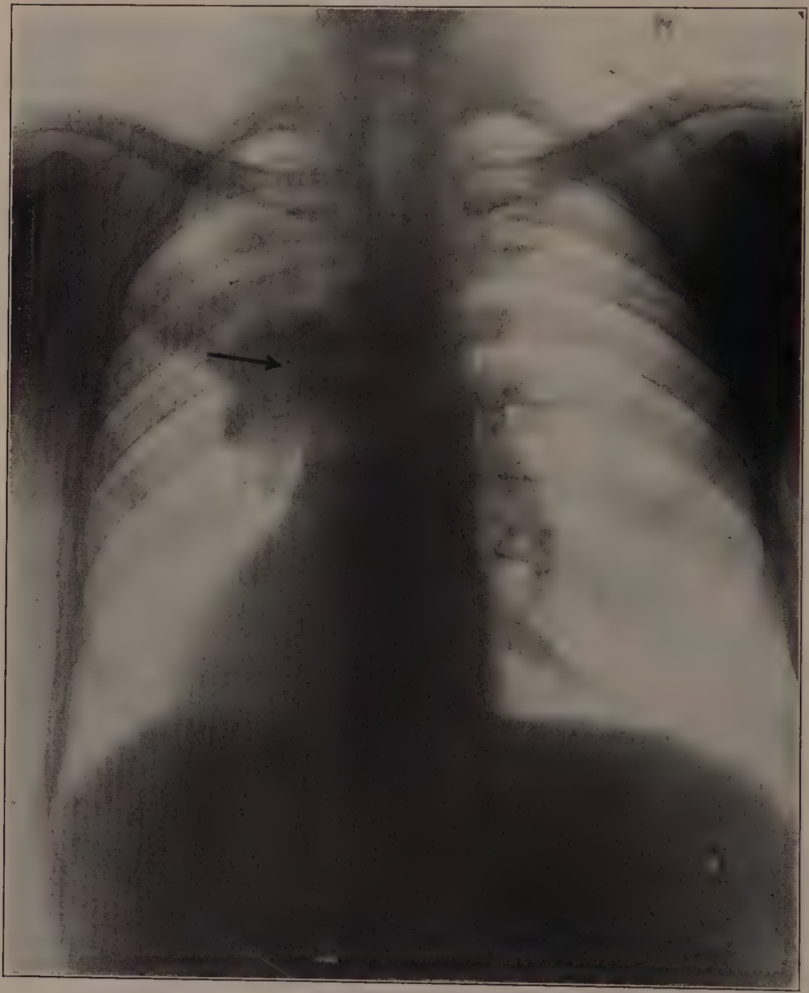

Fig. 292.—Radiograph of carcinoma of the lung. Arrow indicates area of density caused by the tumor.

MICOTIC INFECTIONS OF THE RESPIRATORY TRACT

The lung and bronchi may be invaded by yeasts, molds, or fungi, among the most common being the streptothrix group, the blastomyces and aspergillus fungoides.

Infections with these organisms give rise to no characteristic physical signs. The diagnosis is made by finding evidence of bronchitis or pulmonary consolidation, together with the presence of the organisms in the sputum.

MEDIASTINAL TUMORS

Clinical Pathology.—A substernal thyroid gland or a persistent thymus may by simple enlargement, adenomata, or malignancy produce tumors in the mediastinum.

Sarcoma and carcinoma may appear as primary growths or as metastatic tumors in the mediastinal lymph glands. Hodgkin's disease, the leucemias and tuberculosis may also attack the mediastinal lymph glands, producing tumors of considerable size. Dermoid cyst of the mediastinum is not uncommon. Aneurysm of the arch of the aorta is the most common cause of mediastinal tumor. The exact nature of a mediastinal tumor is often difficult to determine, the final diagnosis often depending on a histologic study of the mass removed surgically or at autopsy.

Physical Signs.—*Inspection* may reveal tumor masses extending into the suprasternal notch or beyond the lateral borders of the sternum. The sternum may be eroded, allowing the tumor masses to present in the upper sternal region, and the trachea may be displaced. If due to aneurysm, or if in contact with the aortic arch, the tumor may pulsate. Pressure upon the superior or inferior vena cava may cause distention of neck veins, veins of the chest, or edema of neck, chest or extremities.

Palpation confirms inspection. The tumor may be smooth in the cause of aneurysm or dermoid cyst, and nodular in the malignant tumors, Hodgkin's disease or the leucemias. Axillary glands may be palpable.

Tracheal tug may be noted if an aneurysm is in contact with the right bronchus.

Percussion.—Dullness over the upper portion of the sternum, extending in any direction, may be noted. A triangular area of dullness with base upward is noted at times with enlarged thymus. If the tumor is of sufficient size, hyperresonance, due to encroachment upon the lungs, may result. Large mediastinal tumors may cause downward displacement of the heart dullness.

Auscultation.—Aneurysm may be associated with a systolic murmur transmitted into the vessels of the neck, and a diastolic shock sound. A solid tumor pressing on the aorta may produce a similar murmur but no shock sound. Pressure on the trachea produces harsh breath sounds sometimes associated with a crowing respiratory murmur.

X-ray.—The area of density is best shown with the patient in the oblique position. Encroachment on the trachea can also be best demonstrated in this position, both on the film and by fluoroscopic examination. The densities of malignant disease, leucemias or Hodgkin's disease are usually irregular. The density of enlarged thymus is often triangular, while that of aneurysm is frequently globular and smooth. Dermoid cyst may produce a globular or irregular shadow. The fluoroscope may reveal the pulsation of aneurysm. Dermoid cyst may show teeth or bone shadows imbedded in the density. Should pressure on the vena cava and lymphatics be sufficient to produce hydrothorax the characteristic x-ray picture will be noted.

CHAPTER XXXIX

DISEASES OF THE PLEURA

PLEURISY (PLEURITIS)

Pleurisy, pleuritis, or inflammation of the pleura, occurs as a primary and as a secondary disease. Primary cases of the disease are frequently attributed by patients to exposure to the elements or to sudden chilling of the body, but in reality they are due to tuberculosis of the pleura in the vast majority of instances. The only nontuberculous cause of primary or idiopathic pleurisy which is worthy of serious consideration is traumatism of the thorax, which readily damages the delicate pleural membrane.

Secondary pleurisy is of far greater frequency than is the primary form of the disease. This type of pleuritis arises as a consequence of extension of disease of contiguous structures, notably of the lung, mediastinum or pericardium; or it develops as a blood-borne infection during the course of general septicemia and pyemia.

The infective agent in pleurisy varies with the type which the disease assumes. Approximately 80 per cent of primary idiopathic pleurisy is induced by infection with the tubercle bacillus. Failure in such cases to demonstrate the bacillus in the pleural exudate or effusion does not exclude a tuberculous origin of the disease, but rather calls for guinea pig inoculation, which frequently proves positive. In the case of secondary pleurisy, on the contrary, in the absence of tuberculosis of the lungs, the pneumococcus is the most productive cause of the disease. It has been estimated that approximately 75 per cent of the cases developing in young children are due to the pneumococcus; and the effusion is usually of the purulent variety. In purulent pleurisy in the adult, again, streptococci, hemolytic and nonhemolytic, are cultured with the greatest frequency from the effusion. Other organisms which may be associated with the streptococcus or present in pure culture comprise various strains of the staphylococcus, the typhoid bacillus, colon bacillus, influenza bacillus, Friedländer's bacillus, and B. diptheriæ.

In accordance with the pathologic findings and the clinical course of the disease, several clinical types of pleurisy are recognized; namely, *acute fibrinous pleurisy; serofibrinous pleurisy,* or *pleurisy with effusion; purulent pleurisy,* or *empyema; local pleurisy;* and *chronic adhesive pleurisy.*

ACUTE FIBRINOUS PLEURISY

(Acute Plastic Pleurisy; Pleuritis Sicca)

Clinical Pathology.—In acute fibrinous pleurisy, pleuritis sicca, or acute plastic inflammation of the pleural membrane, the pathologic changes usually involve the lateral and anterior portions of the pleura, in which site it may be limited to a small area of the visceral pleura, or it may involve a large area of the visceral and parietal membranes. Likewise, the visceral pleura investing the posterior aspect of the pulmonary apex is frequently involved in the disease.

Under the influence of inflammatory change the surface of the pleura in the area of disease becomes dull and lusterless, with a granular, irregular surface. The membrane exhibits a variable degree of thickening; and its surface is clothed with one or more layers of fibrinous exudate. During the movements of respiration the exudate is prone to be rolled into folds upon the surface of the pleura, or it may be thrown up into more or less exuberant masses. A variable amount of clear or cloudy fluid exudes from the inflamed pleura, which, however, never equals the degree of effusion which occurs in serofibrinous pleurisy.

Microscopically, the pleura presents degeneration and desquamation of the surface endothelium at the site of the inflammation, patches of the pleura being found entirely devoid of endothelial covering. At the same time, the subserous connective tissue layer of the membrane is edematous, and exhibits a variable degree of leucocytic infiltration.

Upon microscopic examination of the exudate attending the pleural inflammation, it is found to contain fibrin, serum, and a variable number of pus cells.

Under favorable circumstances acute fibrinous pleurisy undergoes spontaneous resorption, the pleura being restored to a normal state. In more extensive involvement of the pleura, however, the areas of endothelial denudation become united by fibrous adhesions between the visceral and the parietal pleura.

These adhesions may be few in number and result in little embarrassment of pulmonary function, or a large portion of the membranes may become firmly adherent, resulting in a state of chronic adhesive pleurisy.

The onset of acute fibrinous pleurisy is usually acute with pain in the lower anterolateral region of the thorax, or with a dull ache in the upper interscapular region when the apical pleura is involved posteriorly. There is usually a hacking cough, which is not productive, and which serves to aggravate the pain of the inflamed pleura. The disease is attended by a moderate rise of temperature, and acceleration of the pulse, which is usually in proportion to the degree of pyrexia.

Certain cases of the disease pursue a latent course, without pain or other localizing symptoms, and the disease is not detected during the course of the malady which it accompanies. Such cases, however, frequently result in pleural thickening or adhesions.

Physical Signs.—*Inspection.*—In acute fibrinous pleurisy the respiratory movement of the thorax is usually restricted as a result of the stabbing pain which is induced by deep inspiration. As a result the diaphragmatic shadow is commonly absent upon the side of the disease. The trunk is usually inclined toward the side of the disease, with slight dropping of the corresponding shoulder. Changes of posture commonly serve to aggravate the pleural pain.

Palpation.—Palpation of the anterolateral region of the thorax in isolated cases will reveal the presence of pleural friction fremitus over the site of the disease, but this sign is more frequently absent than present.* Vocal fremitus seldom presents any variation in acute plastic pleurisy; but, if pleural thickening is extreme, the intensity of the vocal vibrations is diminished over the area of thickened pleura.

Finger-tip palpation serves to confirm the unilateral deficiency of expansion upon the side of the disease; and palpation of the intercostal spaces over the disease reveals the presence of local tenderness.

Percussion.—The note which is elicited upon percussion of the thorax over an area of acute fibrinous pleurisy in an initial attack of the disease betrays no departure from normal pulmonary resonance; but, in the case of repeated attacks, there may be sufficient pleural thickening to cause moderate impairment of resonance upon the side of the disease. Percussion of the inferior pulmonary borders during inspiration yields a pertinent sign of the disease in the limitation of the respiratory excursion of the diseased lung. Pain is not infrequently elicited upon percussion in this disease.

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*Special technic for eliciting friction rub is described in Part I, page 280.

Auscultation.—Auscultation over the site of the disease reveals the pathognomonic sign of the disease, the pleural friction sound; and, in suitably placed lesions, pleuropericardial friction.* The vesicular murmur is retained, but its intensity is diminished upon the side of the disease as a result of the pain which attends full inspiration. In cases of recurrent pleurisy, which are attended by considerable degrees of pleural thickening, the breath sounds are diminished in intensity over the distribution of the pleural disease.

As a rule, the intensity of the transmitted voice is not modified in acute fibrinous pleurisy; but, in the presence of excessive pleural thickening, vocal resonance may be diminished over the distribution of the disease.

Diagnosis.—The pleural friction sound, when it is elicited, is pathognomonic of acute fibrinous pleurisy. The disease is also attended by moderate fever and by darting, stabbing pain, which is most commonly referred to the region of the axilla or the nipple, and which is accentuated by coughing and by deep inspirations, signs which are not pathognomonic of the disease.

Differential Diagnosis.—*Pleurodynia* is attended by pain in the side, but the pain of this disease is continuous and is aggravated by movement of the trunk as well as by the movements of respiration. Moreover, there is absence of the friction sound and the localization of the pain is not as distinct and clear-cut as it is in pleurisy, the pain occasionally ceasing upon one side of the thorax to appear upon the opposite side of the chest. Pleurodynia is not attended by febrile movement.

Intercostal neuralgia is characterized by sharp, paroxysmal pain over the distribution of the nerve trunks. In this condition there are points of tenderness upon palpation over the points of exit of the nerves upon the lateral and anterior chest walls. Developing as the disease does most frequently upon a neurotic basis, pleural friction and fever are absent in intercostal neuralgia.

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*Special technic for eliciting audible friction rub is described in Part I, page 425.

SEROFIBRINOUS PLEURISY

(Pleurisy with Effusion; Pleuritis Exudativa)

Clinical Pathology.—Serofibrinous pleurisy, pleurisy with effusion, or pleuritis exudativa, represents the progressive development of acute fibrinous pleurisy, when this disease is not early aborted. In this form of pleurisy, in addition to the formation of a fibrinous exudate upon the surface of the inflamed pleura, there is poured forth a more or less abundant serous or serosanguineous effusion, which fills the pleural cavity to a variable extent.

With the development of the effusion the fluid gravitates toward the dependent portions of the pleural cavity, and it mounts progressively higher with the progress of the disease. In certain instances the effusion becomes so extreme that it attains the level of the clavicle.

The inferior border of the lung upon the side of the disease early becomes atelectatic; and, in the case of a large effusion, the lung is crowded into the superior and posterior portions of the pleural cavity, becoming relatively airless. In the case of effusions which persist over a considerable period, fibrosis of the lung ensues; and the organ becomes bound to the chest wall by dense adhesions in the region of the pulmonary apex.

In the presence of a large unilateral pleural effusion the mediastinum is displaced toward the opposite side of the thorax, resulting in displacement of the heart, lateral displacement of the trachea, and the development of paravertebral dullness upon the opposite side of the thorax, *Grocco's triangle.* At the same time, the diaphragm is displaced downward upon the side of the effusion.

In the presence of an extensive unilateral pleural effusion the opposite lung is the seat of compensatory emphysema.

With spontaneous resorption or surgical evacuation of the effusion, fibrous adhesions are prone to form between the visceral and the parietal pleura. These adhesions vary in distribution in individual cases. In certain instances adhesions are few in number; in other cases they are universally distributed over the pleura except for a pocket here and there; while in still other instances they serve to obliterate the pleural sac entirely, resulting in chronic adhesive pleurisy. When a patient with numerous adhesions is the subject of a second attack of serofibrinous pleurisy, only the nonadherent portions of the membranes are involved, with the induction of a loculated, sacculated, or encysted pleurisy.

Cases of serofibrinous pleurisy of moderate degree frequently undergo spontaneous resorption. In other instances the effusion becomes purulent; and in still other cases the disease remains stationary for a prolonged period without essential change in the character of the effusion, constituting *chronic serofibrinous pleurisy.*

In the case of a purely serous effusion the pleural fluid is straw-colored, with a specific gravity of approximately 1.020, and contains flocculi of fibrin, desquamated endothelial cells, leucocytes, erythrocytes, and at times, bacteria. The fluid commonly contains from 4 to 5 per cent of albumin; and upon standing spontaneous coagulation is sometimes observed. In other instances the effusion has imparted to it a red or a brownish tinge, arising from admixture with blood. Again, in the case of pleural effusions attending pulmonary tuberculosis, the fluid frequently has a milky, semitransparent appearance, constituting the chyliform effusion of this disease.

The onset of serofibrinous pleurisy may be abrupt, with pain in the side, coinciding with the initial acute fibrinous inflammation of the pleura, or the disease may develop insidiously during the course of phthisis or one of the acute infections of the bronchopulmonary system. With the development of the effusion the initial pain usually undergoes amelioration. In certain instances pleural effusion develops very rapidly, and is attended by marked dyspnea and urgent distress upon the part of the patient.

The disease is usually attended by febrile movement of moderate degree, particularly during the early period of the illness. The pulse is accelerated, but not out of proportion to the degree of pyrexia, save in cases of tuberculous origin. In the presence of massive pleural effusion the pulse sometimes assumes the paradoxical type.

The prognosis of serofibrinous pleurisy is influenced largely by the underlying cause of the disease, and by the character and amount of the effusion. In the absence of phthisis or malignant disease of the lung, recovery is the rule, usually with a variable degree of pleural thickening or adhesions. The more fibrinous the primary exudate, the greater is the liability to extensive adhesions and permanent crippling of the lung upon the side of the disease. In pleural effusions associated with phthisis the outlook is always grave, and frequently the development of effusion is the first intimation of the presence of pulmonary tuberculosis.

The course of serofibrinous pleurisy is variable, depending largely upon the underlying etiologic agent of the disease. Simple serofibrinous effusions frequently subside in two to three weeks; but the disease in certain instances pursues a lengthy course without apparent reason.

Physical Signs.—*Inspection.*—In serofibrinous pleurisy, with the establishment of effusion the respirations are moderately accelerated as a result of compression of the lung by the fluid, with consequent diminution of the air space upon the side of the disease. In the absence of a very large effusion, however, dyspnea is never extreme, and cyanosis is seldom to be noted.

Depending upon the degree of effusion, the expansion of the thorax is restricted or abolished upon the side of the disease. The deficiency of expansion upon the diseased side is further accentuated by the vicarious expansion of the opposite lung, which is the seat of compensatory emphysema. With all extensive effusions mensuration of the thorax will reveal enlargement upon the side of the disease; and the intercostal spaces are commonly obliterated. Actual bulging of the interspaces, so frequent in empyema, is seldom to be observed in serofibrinous pleurisy. Litten's diaphragmatic shadow is absent upon the side of the effusion.

Unilateral restriction of expansion associated with enlargement of the affected side immediately suggests the pleura as the probable seat of disease rather than the lung, as pulmonary disease resulting in such limitation of expansion is commonly attended by retraction of the affected side of the thorax.

In effusion involving the right pleura the cardiac impulse is displaced toward the left as a result of shifting of the mediastinum, and it is occasionally elevated to the fourth interspace. It is not infrequently localized in the left midclavicular line; and, in extreme cases, it may occupy the left axillary region. In the case of an extensive left-sided effusion, on the other hand, the cardiac impulse is displaced toward the right, and the cardiac apex often occupies a position behind the sternum. In the case of large effusions the apex-beat sometimes is visible to the right of this bone in the third or fourth intercostal space.

The decubitus of the patient in serofibrinous pleurisy is not infrequently suggestive of the disease. During the incipient stage of acute fibrinous pleurisy, prior to the pouring out of the effusion, the patient is prone to lie upon the sound side in the effort to protect the sensitive pleura from pressure; whereas, after the effusion is established, he usually lies upon the side of the disease in order to facilitate the full expansion of the sound lung.

The shoulder upon the side of a large pleural effusion occupies a slightly higher level than does its fellow. Similarly, the nipple and the scapula upon the side of the disease are farther from the median line than upon the opposite side of the thorax.

Dilatation of the pupil upon the side of the effusion, arising from irritation of the intrathoracic sympathetic fibers, is frequently to be noted.

Palpation.—Finger-tip palpation of the two sides of the thorax during respiration is sometimes of advantage in estimating the relative expansion of the two sides of the thorax in cases of moderate effusion. In the presence of large effusions, however, the disproportion between the two sides is so evident as to be readily detected by inspection.

The intensity of vocal fremitus varies according to the degree of effusion and the state of the subjacent lung. In the presence of a moderate effusion which does not fill the pleural sac, vocal fremitus is apt to show slight exaggeration above the level of the fluid, while over the area corresponding to the effusion it is impaired in intensity or is abolished. Posteriorly, near the vertebral column, over the area which is occupied by the compressed lung, vocal fremitus at the height of the effusion is usually exaggerated. In the interpretation of variations in the intensity of vocal fremitus in this disease it should be borne in mind that dense pleural adhesions traversing a pleural effusion will and do transmit the vibrations to the palpating hand, despite the presence of fluid in the pleural sac. Similarly, dense consolidation in the subjacent lung modifies to a degree variations in vocal fremitus over serous effusions.

In the case of a large effusion involving the right pleura, palpation of the upper right quadrant of the abdomen frequently reveals the inferior border of the liver at an abnormally low level below the right costal margin.

Percussion.—During the incipient stage of serofibrinous pleurisy the percussion note is unchanged. In the further evolution of the disease, however, as the effusion develops, there is a progressive impairment of vesicular resonance, finally amounting to flatness over the distribution of the effusion.

Percussion of the thorax immediately above the level of the effusion elicits Skodaic resonance.

The line representing the upper limit of a pleural effusion, as defined by percussion of the thorax, is not horizontal. In the case of a moderate effusion, with the patient in the upright posture, Ellis' line of flatness, indicating the upper limit of the fluid, commences relatively low upon the dorsal aspect of the thorax near the vertebral column and proceeds upward and forward to the axillary region, whence it presents a gradual decline upon the anterolateral region of the thorax. In the presence of a massive effusion, on the contrary, the highest point of the area of flatness is located posteriorly adjacent to the vertebral column, and the superior limit of flatness slopes gradually downward as the axillary region is approached.

Grocco's triangle of paravertebral dullness is demonstrable in most cases of serofibrinous pleurisy. This triangular area, with a width of one to two inches, with its apex directed upward, occupies the side of the thorax opposite the effusion at the level of the eleventh and twelfth ribs. It is probably due to displacement of the mediastinal structures by the pressure of the effusion.

In some cases when the patient lies on the affected side the triangle is diminished, and when lying on the unaffected side it is increased. Purulent effusions are said to produce a larger triangle than serous effusions.

In right-sided effusion the dullness of the fluid blends anteriorly and laterally with the dullness of the liver; whereas an effusion of the left side encroaches upon the tympany of Traube's semilunar space. In serofibrinous pleurisy it is occasionally possible to detect movable dullness upon change of posture. While not always present in this disease, movable dullness when elicited is an infallible sign of fluid in the pleural cavity.

In right-sided effusion cardiac dullness is increased to the left, while in left-sided effusion the dullness is increased to the right of the sternum.

Forcible percussion over the upper portion of the lung, above the level of the effusion, occasionally elicits a cracked-pot sound, produced by the sudden forcible expulsion of air from the relaxed lung. Similarly, upon strong percussion over the infraclavicular region in the presence of large effusions, Williams' tracheal tone may sometimes be elicited.

During absorption of the effusion the dullness gradually is superseded by normal vesicular resonance, save at the bases posteriorly, where the resonance is apt to remain impaired for a long period. Areas of impaired resonance elsewhere point to areas of pleural thickening or to encysted fluid.

Auscultation.—In the incipient stage of the disease, prior to the development of effusion, a pleural friction sound is frequently audible upon auscultation. It is usually best detected in the lower axillary or mammary region. The friction sound usually though not invariably disappears at the height of the effusion, to become once more audible with the absorption of the effusion. When the portion of the pleura which overlies the pericardium is involved, a pleuropericardial friction sound is audible.

The vesicular murmur is abolished over the area of the thorax which overlies the effusion, while above the effusion. the respiratory sounds are puerile or bronchovesicular. While this statement as a rule holds true, yet in very large effusions there is occasionally distant bronchial breathing over the effusion, due to the dense compression of the lung by the fluid which occupies the pleural cavity. The respiratory murmur over the sound lung is exaggerated as a result of compensatory emphysema.

Vocal resonance over the effusion is abolished, unless a patch of pleura be bound to the chest wall by adhesions, in which event the resonance is of approximately normal intensity in the area in question. Upon auscultation just above the level of the effusion during phonation, particularly near the angle of the scapula posteriorly, egophony is frequently elicited.

Upon auscultation of the heart, the sounds are often rather diffusely audible, owing to cardiac displacement. The pulmonic sound is usually accentuated and a systolic murmur may be audible, which is produced by traction upon the vessels by the cardiac displacement.

Mensuration.—Mensuration of the thorax reveals an increase in the circumference of the diseased side of from one-half to one inch. In interpreting the findings of mensuration allowance must be made for the fact that the right half of the thorax is normally larger than is the left side.

X-ray reveals a shadow over the dependent portion of the pleural sac, usually with an irregularly curved superior margin. The density of the shadow varies somewhat with the quality of the effusion, but is little affected by its character. With the aid of the fluoroscope the shadow may sometimes be seen to shift with changes in the position of the patient.

Diagnosis.—The cardinal signs pointing to serofibrinous pleurisy comprise deficient thoracic expansion combined with unilateral enlargement of the thorax; an initial friction sound which disappears with the advent of the effusion; absence of vocal fremitus over the lower portion of the thorax; flatness over the distribution of the fluid, with a paravertebral triangle of dullness on the opposite side; Skodaic resonance above the level of the effusion; absence of respiratory sounds over the effusion with

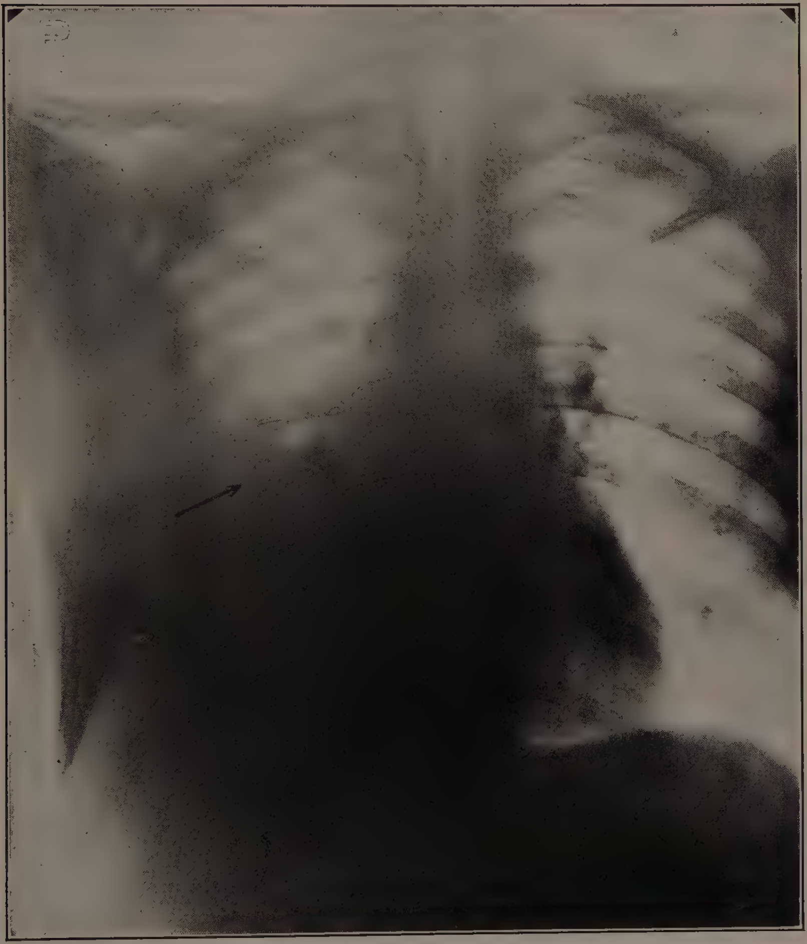

Fig. 293.—Radiograph of serofibrinous pleurisy. Note irregular upper border of density as indicated by arrow.

the presence of puerile, bronchovesicular, or bronchial sounds, sometimes attended by râles, above the level of the fluid; signs of visceral displacement; and certain special phenomena, such as bronchophony and egophony.

The presence of effusion and its character are proved by exploratory puncture of the pleura. However, puncture does not in every positive case reveal the presence of fluid. Even if the needle is inserted in an area of flatness, it may penetrate a region where the thickened pleura is adherent to the chest wall and so fail to secure fluid. Cases of encysted fluid frequently require multiple punctures in order to confirm the diagnosis.

X-ray of the thorax is of distinct aid in diagnosis, especially in the cases of loculated or encysted pleurisy.*

Differential Diagnosis.—Serofibrinous pleurisy is occasionally differentiated from lobar pneumonia with difficulty. The several points of differentiation between these diseases have been enumerated under lobar pneumonia.

From extensive *pericardial effusion,* serofibrinous pleurisy is sometimes differentiated with difficulty, particularly in the cases of effusion into the left pleural cavity. But in pericardial effusion the base of the lung yields resonance instead of flatness; there is Skodaic resonance over the adjacent portion of the lung which is compressed by the effusion; the cardiac impulse is not displaced to the right; the heart sounds are feeble; the pulse is apt to be of the pulsus paradoxus type, trailing off toward full inspiration; and the degree of dyspnea is out of proportion to the extent of the effusion. Moreover, in pericardial effusion the area of dullness occupies the precordia and is pear-shaped with the base resting upon the diaphragm.

Unilateral hydrothorax presents physical signs which are largely identical with those of serofibrinous pleurisy. But in hydrothorax there is absence of the initial friction sound; there is no primary stitch in the side, or fever, but instead there is a history of heart disease, or of nephritis. Moreover, hydrothorax is frequently attended by edema of dependent portions of the body.

Ellis' curve is not so pronounced in hydrothorax and upon exploratory puncture the fluid of hydrothorax is more serous, and is of lower specific gravity; it contains less than three per cent of albumin; and it never coagulates spontaneously upon standing. In hydrothorax movable dullness is usually readily elicited, as the fluid of hydrothorax readily shifts with change of posture, while in pleurisy with effusion this sign is elicited with difficulty if, indeed, it is elicited at all.

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*Technic of exploratory puncture is described in Part I, page 480.

Intrathoracic neoplasms may simulate serofibrinous pleurisy. They frequently produce displacement of the cardiac impulse, as does pleurisy with effusion. The dullness of neoplasm, however, occupies the central portion of the thorax and is of minor extent, and it is surrounded by a zone of compressed lung, yielding Skodaic resonance. Vocal fremitus and vocal resonance are increased rather than diminished. The breath sounds are frequently suppressed, and at other times distinctly bronchial. Pulmonary neoplasm often coexists with moderate pleural effusion. Malignant neoplasms are prone to produce glandular enlargement in the supraclavicular region, and are eventually attended by emaciation and cachexia. These growths also cause enlargement of the mediastinal glands, producing in this wise pressure paralysis of the recurrent laryngeal nerves. Moreover, in the case of neoplasm, the physical signs are not influenced by change of posture.

Hepatic enlargement from abscess, echinococcus cyst, or enlargement from subphrenic abscess may simulate pleurisy with effusion. But the upper limit of dullness is immovable; it presents a convex outline directed upward; and it frequently exhibits a friction sound in the midst of the area of dullness, which would not be true if the pleural surfaces were separated by effusion. These conditions are not infrequently attended by moderate pleural effusion and hence coexistent therewith. Upon exploratory puncture the pus from an hepatic abscess may perhaps contain liver cells or bile, and occasionally amebæ. Grocco's sign is absent in hepatic enlargement. Occasionally hydatid fremitus may be elicited upon percussion over an echinococcus cyst of the liver.

Pneumothorax produces unilateral bulging of the thorax with immobilization; but the percussion note is hyperresonant or tympanitic; and the disease is attended by characteristic physical signs.

PURULENT PLEURISY

(Empyema)

Clinical Pathology.—While it is possible for a serofibrinous pleurisy to become purulent as a result of secondary infection with pyogenic organisms, in the vast majority of cases purulent pleurisy, or empyema, arises as an independent disease, with no relation to a preexisting acute fibrinous or serofibrinous pleurisy.

Purulent pleurisy is noted in rare instances as a primary disease, chiefly in young subjects from infection with the pneumococcus; but in the majority of cases it is secondary to disease or injury of the lung or thorax.

Empyema complicates many acute diseases, notably scarlatina, lobar and lobular pneumonia, abscess and gangrene of the lung, and it is not infrequently observed during the course of pulmonary tuberculosis. The disease may be caused by penetration of the chest wall by a fractured rib or by other perforating wound. Similarly, perforation of the diaphragm by subphrenic abscess is frequently followed by empyema; and carcinoma of the esophagus is prone to penetrate the pleura and to initiate a purulent pleurisy.

The pneumococcus is responsible for the greater number of cases of empyema, the disease occurring as a complication of lobar pneumonia, or developing independently of this disease. A considerable number of cases of chronic phthisis are attended by purulent effusion into the pleura. The precise rôle of the tubercle bacillus in the induction of these effusions has not been determined. As a rule, mixed infection with pyogenic micrococci can be demonstrated in such effusions. In other instances of empyema complicating phthisis the purulent effusion is sterile upon culture, a finding which suggests a tuberculous origin of the disease. Other organisms which are frequently causative of purulent pleurisy comprise the streptococcus, staphylococcus, bacillus influenzæ, colon bacillus, and the bacillus aerogenes capsulatus.

The character of the effusion varies with the duration of the disease and with the type of infecting organism. Early in the course of empyema exploratory puncture may reveal a fluid which is merely turbid from a moderate admixture with leucocytes. With the further evolution of the disease the effusion becomes thicker and more or less creamy in appearance. As a rule, pneumococcic effusions are thick, heavily loaded with pus cells, with a variable admixture of fibrin, and the odor is seldom fetid. Effusions induced by streptococci, on the other hand, are usually thin, with a moderate turbidity, the cellular elements tending to settle toward the base of the pleural sac. In certain cases the effusion becomes infected with saprophytic bacteria, resulting in a fluid of exceedingly foul odor. In other instances gas-forming organisms are present, and convert an empyema into a pyopneumothorax.

In empyema the pleural cavity contains a variable amount of purulent effusion, which may be free in the sac or may be encysted through the formation of adhesions. The solid constituents of the effusion gravitate toward the dependent portions of the pleural sac, while the upper strata consist of more or less turbid fluid. This factor may be productive of an error in diagnosing the type of effusion; because, if the aspirating needle is entered above the level of the distinctly purulent stratum, only turbid fluid may be withdrawn. At the height of the disease, however, the fluid is uniformly thick and purulent, save in certain cases of streptococcic origin.

As the effusion mounts in the pleural cavity, the lung is compressed to a variable degree, in extreme cases occupying a small area in the superior portion of the pleural cavity near the vertebral column. In purulent pleurisy adhesions form with far greater rapidity than is the case in serofibrinous effusions. The newly formed adhesions, moreover, undergo organization rapidly, and in all cases of extensive effusion there is permanent crippling of the pulmonary function. In an effusion of moderate volume fibrous adhesions between the visceral and the parietal pleura frequently wall off the pus, forming in this wise a local abscess cavity.

Empyema is constantly attended by retrogressive changes in the lung. With the establishment of adhesions the subpleural lymphatics early become involved in the inflammation, and an interstitial pneumonia of pleurogenous origin extends for a variable distance into the lung. Moreover, the compression of the lung induces atelectasis of the lower lobe, which becomes splenized, relatively airless, and finally cirrhotic and of dark brown color.

In extensive unilateral empyema the heart is displaced toward the opposite side of the thorax; and the impact of this organ against the fluid during systole results in a local pulsation of the thoracic wall, *pulsating empyema.* Similarly, the diaphragm is commonly displaced downward upon the side of the disease, with consequent depression of the liver or spleen.

In compensation for the crippling of the lung upon the side of the disease in unilateral cases of empyema the opposite lung is the seat of compensatory emphysema.

Every large empyema produces permanent deformity of the thorax. As the effusion accumulates the side of the chest corresponding to the disease is enlarged; and the intercostal spaces are obliterated or actually present local bulging, from the pressure which is exerted upon the intercostal muscles and their investing fascia. Depression of the diaphragm results in restriction of the excursion of this muscle and an increase in the vertical diameter of the thorax. Following absorption or surgical evacuation of the effusion, the intercostal spaces upon the side of the disease are retracted; the thoracic wall undergoes partial collapse; the shoulder droops to a variable degree; and the vertebral column presents varying grades of scoliosis.

A purulent pleural effusion is apt to burrow beneath the costal pleura and to point subcutaneously, constituting *empyema necessitatis;* and spontaneous evacuation of the effusion may occur unless the pressure is relieved by prompt aspiration. In other instances the effusion perforates the visceral pleura and discharges into a bronchus, with the induction of pyopneumothorax. Perforation of the esophagus has been noted. Also in certain cases the purulent effusion perforates the diaphragm; and, passing downward along the vertebral column and psoas muscle, points in the thigh.

Empyema may begin abruptly, with chill followed by a rise of temperature, simulating to a certain degree the onset of lobar pneumonia. More frequently, however, the disease develops insidiously during the course of phthisis or an acute pulmonary infection.

The disease pursues a lengthy course, sometimes interrupted by transient periods of amelioration of the signs and symptoms, but with a progressive impairment of physical vigor and multiplying signs of septic intoxication. Empyema is attended by an absolute increase in the leucocytes, the count in grave cases sometimes approximating 100,000 cells per c.mm. While it is possible for purulent effusions of moderate degree to undergo spontaneous absorption, this change seldom occurs; and, in the absence of surgical intervention, the prognosis is always grave.

Physical Signs.—*Inspection.*—The physical manifestations of purulent pleurisy which are noted upon inspection are, in the main, very similar to those of serofibrinous pleurisy with effusion; but in empyema the side of the thorax corresponding to the disease commonly presents a greater degree of bulging, and there is more apt to be bulging of the intercostal spaces, particularly over the lower region of the thorax. Edema of the chest wall is in evidence when the parietal pleura is extensively involved; and in empyema necessitatis there is a visible elevation of a localized region of the thoracic wall, with discoloration of the integument when rupture is imminent.

The cardiac impulse is displaced toward the opposite side of the thorax in unilateral effusions; and, in the case of a left-sided effusion, it may be invisible as a result of displacement of the cardiac apex behind the sternum. Depression of the diaphragm may produce local bulging below the costal arch.

Expansion of the thorax upon the side of the effusion is restricted or abolished, while the opposite side in unilateral empyema expands vicariously as a result of compensatory emphysema.

The subcutaneous veins are sometimes enlarged and tortuous over the lower thorax upon the side of the disease; and the diaphragmatic shadow is abolished upon the affected side. In the presence of pulsating empyema there is to be noted over the lower anterolateral region of the thorax a systolic pulsation, which is synchronous with ventricular systole.

Palpation.—As in the case of serofibrinous effusions, in empyema vocal fremitus is abolished over the distribution of the purulent effusion, while above the level of the fluid, the fremitus is usually exaggerated.

Pulsating empyema yields a palpable systolic pulsation of the chest wall, while in empyema necessitatis the edematous wall frequently pits upon pressure.

Percussion.—Percussion of the thorax over the distribution of a purulent effusion yields flatness, with Skodaic resonance above the level of the fluid. In massive effusions the superior limit of the zone of flatness is quite similar to that attending serofibrinous effusions. In a loculated effusion, on the contrary, the dull or flat area is circumscribed to a limited region of the thorax.

In unilateral effusions percussion of the opposite side of the thorax yields a moderately hyperresonant note, as a result of compensatory emphysema of the subjacent lung. Similarly, Grocco's triangular area of paravertebral dullness is usually more marked in empyema than in serofibrinous pleurisy.

Auscultation.—As in the case of serofibrinous pleurisy with effusion, in empyema the respiratory murmur is commonly abolished over the distribution of the purulent effusion, while it is puerile or bronchovesicular over the distribution of the compressed lung, above the level of the fluid. Frequently the râles of a concomitant bronchitis are to be elicited over the distribution of the lung; and egophony can usually be detected above the effusion during phonation.

In young subjects, in the presence of moderate effusions, distant breath sounds can occasionally be detected over the distribution of the fluid. This finding is to be attributed to coincident consolidation or condensation of the pulmonary tissues.

X-ray film is identical with that of serofibrinous pleurisy.

Diagnosis.—Purulent pleurisy of brief duration rather closely simulates serofibrinous pleurisy in its physical manifestations; but usually in empyema the disproportion between the two sides of the thorax is more remarkable; there is more apt to be intercostal bulging in empyema; and the visceral displacement in purulent pleurisy is more extreme. Likewise, local edema of the thoracic wall points to purulent rather than to serofibrinous pleurisy. Moreover, as a rule, empyema is attended by a greater degree of dyspnea than is serofibrinous effusion, the labored breathing frequently attaining to the degree of orthopnea. The temperature in empyema is usually higher and its course more irregular than in serofibrinous pleurisy.

In the late case of empyema, the emaciation of the patient points to a condition of advanced septic intoxication, and is apt to suggest the diagnosis of chronic phthisis, pulmonary fibrosis, or bronchiectasis.

Resort to the aspirating needle and to the x-rays frequently serves to reveal the true nature of the disease. Similarly, examination of the blood in empyema is very prone to exhibit a leucocytosis, which is frequently absent in the serofibrinous form of the disease.

LOCAL PLEURISY

Under the head of local or circumscribed pleurisy are embraced *diaphragmatic pleurisy; loculated, sacculated, or encysted pleurisy; and interlobar pleurisy.*

LOCULATED, SACCULATED, OR ENCYSTED PLEURISY

Clinical Pathology.—This type of pleurisy may be serofibrinous, but is more frequently purulent. In this form of pleurisy the fluid is circumscribed by adhesions between the visceral and parietal pleuræ into one or more pockets or loculi, which may or may not communicate with one another. While these loculi of effusion may develop in any portion of the pleural cavity, they are most frequently situated in the region between the midaxillary line and the midspinal line, or upon the thoracic aspect of the diaphragm. In these cases the fluid may be bounded by adhesions, the result of a previous attack of pleurisy; or an empyema may become limited and circumscribed by newly formed inflammatory adhesions.

Physical Signs.—The physical signs in this type of pleurisy are few and are frequently atypical. Areas of dullness may be elicited in certain cases; but this is by no means the rule; and vocal fremitus may be clearly transmitted by the adhesions despite the presence of fluid in the pleural cavity. X-ray and the free use of the aspirating needle are the surest means of diagnosis.

INTERLOBAR PLEURISY

Clinical Pathology.—In the evolution of serofibrinous or purulent pleurisy the pleura clothing the interlobar fissures of the lung is also implicated in the inflammation, and not infrequently becomes adherent, enclosing between the two pleural layers a variable quantity of serofibrinous or purulent fluid.

Also in cases of lobar pneumonia and pulmonary tuberculosis an interlobar pleurisy may occur, with adhesions of the pleural surfaces and retention of effusion. Interlobar pleurisy is usually purulent, and in its physical manifestations it frequently closely simulates pulmonary abscess. Such a collection of pus may perforate and discharge into a bronchus and lead to the expectoration of purulent sputum.

Interlobar pleurisy develops with the greatest frequency near the root of the right lung, involving the pleura which dips into the fissure between the upper and middle lobes of the lung.

Physical Signs.—The physical signs of interlobar pleurisy are frequently atypical and confusing. As a rule, there is little or no dullness upon percussion; but in certain cases a zone of dullness corresponding to the course of the fissure between the upper and middle lobes of the right lung can be found, limited above and below by a zone of Skodaic resonance.

Diagnosis.—The x-ray is of aid in the diagnosis. Aspiration is dangerous, as the lung is apt to be infected during withdrawal of the needle. The clinical picture frequently closely simulates that of pulmonary abscess.

CHRONIC ADHESIVE PLEURISY

Clinical Pathology.—Chronic adhesive pleurisy, or chronic plastic pleurisy, arises most frequently as a sequel of serofibrinous or purulent pleurisy; but occasionally it develops as a primary affection of the pleura.

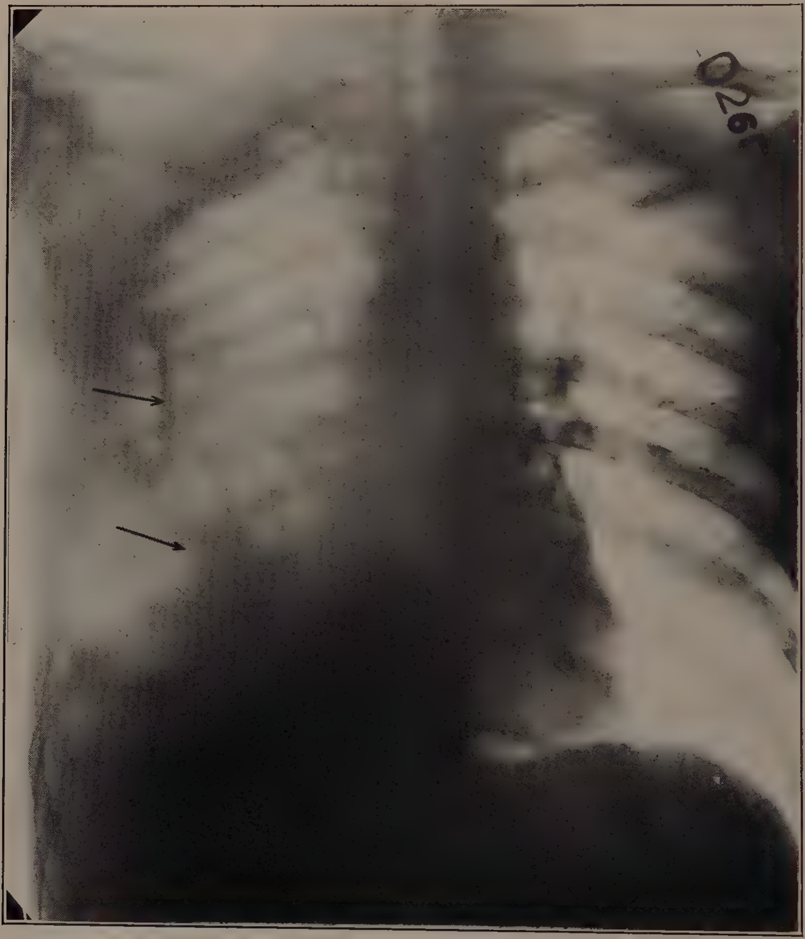

Fig. 294.—Radiograph of chronic adhesive pleurisy. Arrows indicate densities due to bands of adhesions.

Following the spontaneous absorption or surgical evacuation of a pleural effusion, the pleural surface is clothed by an exudate which is rich in fibrinous elements; and, moreover, there are scattered areas of endothelial denudation in which the subjacent connective tissue base of the pleural membrane is exposed. The surfaces of the visceral and the parietal pleura, partially denuded of endothelial covering and clothed with an inflammatory exudate, readily become adherent; and, the fibrinous exudate having undergone organization, the two surfaces become bound together by fibrous adhesions.

The adhesions may be relatively few, or they may be abundant, crippling the lung to a remarkable degree. In extreme cases the pleural membranes may become adherent practically throughout their entire distribution, obliterating the potential pleural cavity upon the side of the disease.

Physical Signs.—The physical signs of chronic adhesive pleurisy vary with the duration of the disease and with the extent of the pleural adhesions, varying from moderate dyspnea to extreme embarrassment of the respiratory function and striking thoracic deformity.

Inspection.—In cases of chronic adhesive pleurisy with moderate adhesions slight dyspnea may be the only sign of the disease, and even this sign may not be in evidence. But in the more extreme grade of the disease, in which numerous adhesions between the visceral and the parietal pleura bind the lung to the thoracic wall, local retraction or absolute immobilization of the corresponding side of the thorax is in evidence. Litten's sign may be diminished or absent.

Palpation.—Upon palpation of the thorax pleural friction fremitus is demonstrable over the sites of pleural roughening and thickening. Palpation is apt to detect minor degrees of expansion and thoracic retraction in cases of moderate adhesions. In cases with considerable thickening of the pleura the intensity of vocal fremitus is diminished.

Percussion.—The percussion note is but little altered in cases of moderate pleural involvement; but the note is strikingly dull and the resistance to the pleximeter is marked in cases with obliteration of extensive portions of the potential pleural cavity. The respiratory excursion of the lung is greatly diminished, occasionally to complete immobilization, upon the side of the disease. The note upon percussion of the opposite lung in the extreme case is hyperresonant as a consequence of compensatory emphysema.

Auscultation.—In chronic adhesive pleurisy the respiratory murmur is very apt to be enfeebled over various areas or indeed in the extreme case over the entire side of the thorax. Over areas of pleural roughening the pleural friction sound is audible;

and very frequently the râles of an associated chronic bronchitis are audible upon the side of the disease.

X-ray shows bands of increased density due to the fibrous adhesions.

Diagnosis.—The diagnosis is made upon the presence of the pleural friction sound, the thoracic deformity and respiratory embarrassment, with a history of previous acute pleurisy.

DIAPHRAGMATIC PLEURISY

Clinical Pathology.—In diaphragmatic pleurisy the inflammation is limited to the parietal pleura which invests the superior surface of the diaphragm and to the visceral pleura which is in contact with it.

The pleural inflammation as a rule assumes the dry, plastic type; but there is sometimes moderate effusion, which may be serous or purulent in character.

Physical Signs.—The physical signs of diaphragmatic pleurisy are slight in comparison with the subjective symptoms, which are unusually severe in their manifestations. There is urgent dyspnea; and the lower region of the thorax is fixed, moving very slightly with respiration. A friction sound can occasionally be elicited over the hepatic area in right pleural inflammation, or over Traube's semilunar space in left-sided inflammation.

There is tenderness upon pressure upon the lower intercostal spaces near the vertebral column, and extreme pain upon pressure over the insertion of the diaphragm at the tenth rib. There is occasionally tenderness upon pressure over the course of the phrenic nerve in the cervical region. Dysphagia is occasionally present, as a result of involvement of the esophageal orifice in the diaphragm; and hiccough and vomiting may accompany left-sided diaphragmatic pleurisy. The vomiting and respiratory movements exaggerate the pain of the disease. Pain is constant in the epigastric region, simulating in this respect acute disease of the abdominal viscera. The diaphragm is fixed, and the respiration is purely costal in type.

Diagnosis.—The diagnosis of diaphragmatic pleurisy rests upon the great severity of the subjective symptoms and the meagerness of physical signs. Gueneau de Mussy states that pain extending from the tenth rib to the ensiform cartilage is pathognomonic of diaphragmatic pleurisy. Andral has noted cases attended by excessive dyspnea and attacks simulating angina pectoris.

HEMOTHORAX

Clinical Pathology.—The collection of blood in the pleural cavity may result from rupture of an aneurysm of one of the larger intrathoracic arteries or from erosion of an intercostal vessel in the presence of pleural disease. Hemothorax also arises as a result of trauma to the chest wall, notably after perforating wounds from missiles or from a fractured rib. Gangrene of the lung may be responsible for the hemorrhage, or it may be a portion of a hemorrhagic diathesis. Rupture of aneurysm of the aorta usually produces hemothorax of the left pleural cavity.

The onset of hemothorax is usually very abrupt, and if one of the larger intrathoracic vessels is the source, it is usually rapidly fatal. In bleeding from an intercostal vessel the bleeding is apt to be slowly continuous for a variable time, after which it spontaneously ceases.

The amount of blood which is extravasated into the pleural cavity is variable. If infection does not occur, following arrest of the hemorrhage, complete absorption may occur; not, however, without leaving pleural adhesions.

Physical Signs.—The initial signs of hemothorax are those of internal hemorrhage; namely, pallor, with dyspnea, rapid pulse, and collapse. Superimposed upon these signs are those of effusion in the pleural cavity; and exploratory puncture reveals the presence of sanguineous fluid.

The *x-ray* film is identical with that of serofibrinous pleurisy.

CHYLOTHORAX

Clinical Pathology.—Effusion of chyle or chyliform fluid into the pleural cavity is only occasionally encountered. The fluid may be derived from rupture of the thoracic duct or may be discharged by transudation from the lacteals. The thoracic duct may be ruptured by trauma to the thorax; or the duct may be obstructed by the pressure of an intrathoracic tumor. Again, a chylous ascites may discharge into the pleural cavity by way of the lymphatics. Or, again, occlusion of the left subclavian vein, into which the thoracic duct empties its contents, may result in chylothorax.

Physical Signs.—The physical signs of chylothorax are essentially those of fluid in the pleural cavity. The nature of the effusion is determined by exploratory puncture of the pleura.

HYDROTHORAX

Clinical Pathology.—Hydrothorax, the collection of a non-inflammatory transudate in the pleural cavity, occurs most frequently in connection with imperfectly compensated cardiac disease and chronic nephritis. Likewise, hydrothorax has been noted during the development of aortic aneurysm, mediastinal neoplasm, and hepatic cirrhosis.

Hydrothorax of cardiac origin usually develops during the course of chronic myocarditis, particularly when stenosis of the mitral valve is present. Under these circumstances the serous effusion is usually unilateral, occupying the right pleural sac. The predilection of this form of the disease for the right pleural sac was ascribed by Baccelli to partial obstruction of the vena azygos major, as this vessel was assumed to be drawn against the root of the lung as a result of depression of the superior vena cava by the enlarged heart. Steele and Stengel attribute the right-sided distribution of the disease to compression of the azygos vein by an enlarged right auricle, while Fetterolf and Landis conclude that the underlying cause is compression of the pulmonary veins at the root of the lung by an enlarged heart, particularly by a dilated right auricle. In bilateral cases of hydrothorax which are attributable to cardiac insufficiency the effusion is always greater upon the right side.

Hydrothorax of renal origin is usually bilateral, and it is uncommon for the effusion to become as extensive as it is in cases of cardiac origin.

The fluid of hydrothorax is a true transudate. It is free from fibrin, is clear, is of light yellow hue, and has a specific gravity below 1.018.

In uncomplicated hydrothorax the pleura exhibits little departure from its normal state. In cases of long standing, however, the surface of the pleura investing the dependent portions of the sac loses its glistening appearance, and is coated with a gray deposit, which is readily removed and which is not fibrinous in character. Occasionally adhesions from a previous acute pleurisy result in a condition of loculated or encysted hydrothorax.

Extensive hydrothorax is attended by visceral displacements which are similar to those which attend inflammatory effusions into the pleura. But, while the lung is compressed by the fluid, it is readily reinflated upon evacuation of the transudate.

Physical Signs.—The physical signs of hydrothorax are essentially those of effusion into the pleural cavity, but the initial friction sound of pleurisy is absent. More commonly limited to the right pleura, the effusion is invariably greater upon the right side.

X-ray film is identical with that of serofibrinous pleurisy.

Diagnosis.—In every case of hydrothorax it is desirable from a prognostic and therapeutic standpoint to determine the cause of the serous effusion. In cases of cardiac origin the orthodiagram commonly reveals an enlargement of the heart in one or more dimensions, and auscultation of the precordia not infrequently reveals the murmurs of valvular lesions.

In cases which are dependent upon renal disease, signs referable to the renal insufficiency are usually in evidence. In the absence of general anasarca, moderate edema of the infraorbital regions as well as of the ankles is frequently demonstrable. Examination of the urine and functional renal tests are frequently of aid in elucidating the provocative lesion of the disease.

X-ray and exploratory puncture confirm the presence and demonstrate the character of the pleural effusion.

PNEUMOTHORAX, HYDRO-, PYO-, AND HEMOPNEUMOTHORAX

Clinical Pathology.—The accumulation of air or gas in the pleural cavity may result from trauma of the thorax, or it may arise as a complication of disease of the lung or of neighboring organs. Rarely the disease develops as an independent affection, with no demonstrable causative lesion in the chest or abdomen, *spontaneous pneumothorax.* Associated with the pneumothorax there is usually a variable quantity of serous fluid, pus, or blood, constituting hydro-, pyo-, or hemo-pneumothorax, respectively.

Penetrating wounds of the chest wall or penetration of the parietal pleura by a fractured rib may be responsible for the induction of pneumothorax. In other instances the disease is caused by perforative lesions of the mediastinum, esophagus, stomach, colon, liver or subdiaphragmatic space. Likewise, the disease has been induced during exploratory puncture or therapeutic aspiration of the pleura.

The most fertile source of pneumothorax, however, is represented by pulmonary disease, tuberculosis of the lung occupying the place of first importance. In a series of 918 cases of pneumothorax Biach found pulmonary tuberculosis responsible for the disease in 77 per cent, gangrene of the lung in 7 per cent, empyema in 5 per cent, emphysema, infarction, thoracentesis, echinococcus cyst and mediastinal disease in 4 per cent, trauma to the thorax in 3 per cent, bronchiectasis and pulmonary abscess in 1 per cent, while in the remaining 3 per cent the cause could not be determined.

In rapidly developing cases of phthisis an area of caseation immediately subjacent to the pleura may perforate this membrane by ulceration, or the visceral pleura may rupture during a severe paroxysm of cough. Similarly, a subpleural tuberculous cavity may by rupture into the pleural cavity establish pneumothorax. Under these conditions the entrance of secondary infection usually results in the development of pyopneumothorax.

The usual site of rupture of the visceral pleura is in the upper lobe, usually between the third and the sixth ribs. The perforation is single in the vast majority of cases, although multiple perforations rarely occur. The accident is prone to occur in portions of the lung which are the seat of rapidly spreading disease rather than in the site of the original focus, since the lung overlying this region is commonly united to the parietal pleura by adhesions. In cases developing in bilateral phthisis pneumothorax more frequently occurs upon the side of secondary involvement. The communicating fistula may remain patent, or the abnormal orifice may subsequently become sealed.

In the presence of empyema the visceral pleura may be penetrated, and a communication established with the bronchial system, in this wise inducing a retrograde pyopneumothorax.

Rarely the development in the pleural cavity of gas-producing organisms, notably the bacillus aerogenes capsulatus and the colon bacillus, results in the establishment of pneumothorax in the absence of perforation of the pleura. In cases of so-called spontaneous pneumothorax it is possible that a previously existing perforation has become obliterated.

Pneumothorax is nearly always a unilateral disease, and the affection involves the two sides with about equal frequency. The cases are most commonly encountered in young adults, and males are attacked more frequently than are females.

According to its distribution, two forms of the disease are recognized; namely, *free* or *diffuse pneumothorax,* and *local* or *circumscribed pneumothorax.* In the absence of pleural adhesions over the inferior portions of the pleura, the entrance of air into the pleural sac results in diffuse pneumothorax, a collection of air of relatively large distribution. In these cases the lung is crowded into the superior portion of the pleural cavity; the diaphragm upon the side of the disease is depressed to a variable degree; and the mediastinum and heart are displaced toward the opposite side. In the presence of numerous adhesions, on the contrary, pleural rupture may result in the formation of local pneumothorax of limited extent, which is attended by little visceral displacement and few clinical symptoms. In 70 per cent of cases circumscribed pneumothorax develops above the third rib, in which site it may readily be mistaken for a pulmonary cavity.

According to the degree of patency of the fistulous communication, Weil has divided pneumothorax into three types: namely, *open pneumothorax,* in which the air during respiration has free ingress and egress through a patulous fistula; *valvular pneumothorax,* in which the orifice is of such character that the air enters the pleural sac during inspiration, but is prevented from being expelled during expiration; and *closed pneumothorax,* in which the pleural perforation has become sealed, preventing communication with the external air.

The onset of pneumothorax varies with the type which the disease assumes and the circumstances under which it develops. Cases of local pneumothorax which develop during the course of advanced phthisis are usually attended by little visceral displacement and few acute symptoms. In the case of diffuse pneumothorax, on the contrary, the onset is usually abrupt with urgent symptoms, such as severe pain in the side, dyspnea amounting to orthopnea or imminent suffocation, cough, and occasionally cyanosis and collapse.

The prognosis varies with the distribution of the disease, and with the state of the bronchopulmonary system. Local pneumothorax of limited distribution exercises little influence upon the course of the disease which it complicates. In the case of diffuse pneumothorax, on the other hand, the patient is apt to succumb to shock at the time of the accident. If he survives, the ultimate outlook varies with the cause of the pneumothorax. Cases complicating phthisis frequently survive only a few weeks, although the condition may become stationary, and the patient may survive for a period of months or years. In purely traumatic cases, in the absence of disease of the lung, the outlook is favorable, the vast majority of the cases terminating in clinical recovery.

Physical Signs.—*Inspection.*—The findings upon inspection in pneumothorax vary with different periods of the disease and with the underlying cause of the malady. At the time of onset of a diffuse pneumothorax the patient is seized with dyspnea which often amounts to orthopnea, and presents signs of shock, notably pallor, sweating, and cold extremities.

In established cases of pneumothorax a different picture is presented. In such a case compensatory changes in the thoracic viscera have occurred, and dyspnea is not always a striking feature of the disease. The state of the thorax upon the side of the disease varies with the degree of pneumothorax and with the presence or absence of fluid in the pleural cavity. As a rule, the chest upon the side of the disease presents a very noticeable enlargement, with restriction of expansion; and, in many instances, complete immobility of the side is noted. In late stages of the disease, associated with purulent effusion, however, the lower region of the affected side may exhibit local retraction from adhesions. In addition to enlargement of the involved side, the intercostal spaces are commonly obliterated. Actual bulging, however, is seldom to be noted in the absence of coincident purulent effusion. In cases of extensive duration the opposite side of the thorax expands vicariously as a result of compensatory emphysema.

In diffuse pneumothorax the cardiac impulse is displaced to a variable distance toward the opposite side of the thorax, unless extensive adhesions of the opposite lung prevent mediastinal shifting. Likewise, downward displacement of the diaphragm upon the side of the disease results in abnormal depression of the liver or the spleen. Litten's diaphragmatic shadow is absent upon the side of the disease.

The decubitus of the patient is not characteristic of the disease. While in numerous instances the subject prefers to lie upon the side of the pneumothorax in order to afford free play to the sound lung, dyspnea may necessitate the upright posture, while in cases complicated by empyema with bronchial fistula peculiar postures may be assumed in order to favor free drainage.

Palpation.—Vocal fremitus is markedly enfeebled or is abolished over the distribution of a diffuse pneumothorax, save in cases where dense pleural adhesions bind a portion of the lung to the chest wall. Finger-tip palpation of the intercostal spaces yields a sensation of increased resistance, which is definitely increased when the superior level of a collection of fluid is reached.

Bilateral palpation of the lower lateral thoracic regions is useful in determining deficiency of expansion of the diseased side; and palpation of the precordia usually reveals displacement of the cardiac impulse from its normal site. Similarly, palpation of the upper quadrants of the abdomen is prone to reveal displacement of the liver or of the spleen upon the side of the disease.

Percussion.—The findings upon percussion in pneumothorax vary with the tension of the air in the pleural cavity, and with the degree of effusion which complicates the disease. In the presence of an extensive collection of air which is under moderate tension the percussion note may be moderately tympanitic and of comparatively low pitch. With collections of gas under considerable pressure the note is of higher pitch and of brief duration, somewhat simulating the tone which is elicited upon percussion over the intestine when it is distended with gas.

In the presence of coincident effusion percussion of the inferior portion of the thorax yields flatness, the note changing abruptly to tympany when the upper border of the fluid is passed. The upper level of fluid, in the absence of adhesions, is a straight line instead of the S-curve noted in serofibrinous pleurisy. With changes in posture of the patient, shifting dullness is elicited. Tympany may extend upward above the clavicle; and, in the absence of effusion, there is similar tympany downward over the hepatic or splenic region.

Pneumothorax with patent bronchial communication frequently yields a cracked-pot sound upon forcible percussion, and occasionally Wintrich's change of note is to be elicited. Likewise, in such cases Biermer's phenomenon, an alteration in the pitch of the percussion note with change in the patient's posture, can sometimes be demonstrated.

In the case of left-sided pneumothorax the area of cardiac dullness is commonly restricted or completely displaced, percussion of the precordia yielding hyperresonance or tympany as a result of shifting of the mediastinum. In right-sided pneumothorax the cardiac dullness is displaced to the left.

Percussion of the upper quadrants of the abdomen is apt to reveal a downward displacement of the liver or spleen upon the side of the disease.

Auscultation.—As a rule, the respiratory sounds over the distribution of a diffuse pneumothorax are very distant or are abolished. In other instances distant amphoric breathing is audible. Over the opposite lung, on the contrary, the respiration is puerile as a result of compensatory emphysema, if the disease has persisted for a considerable period.

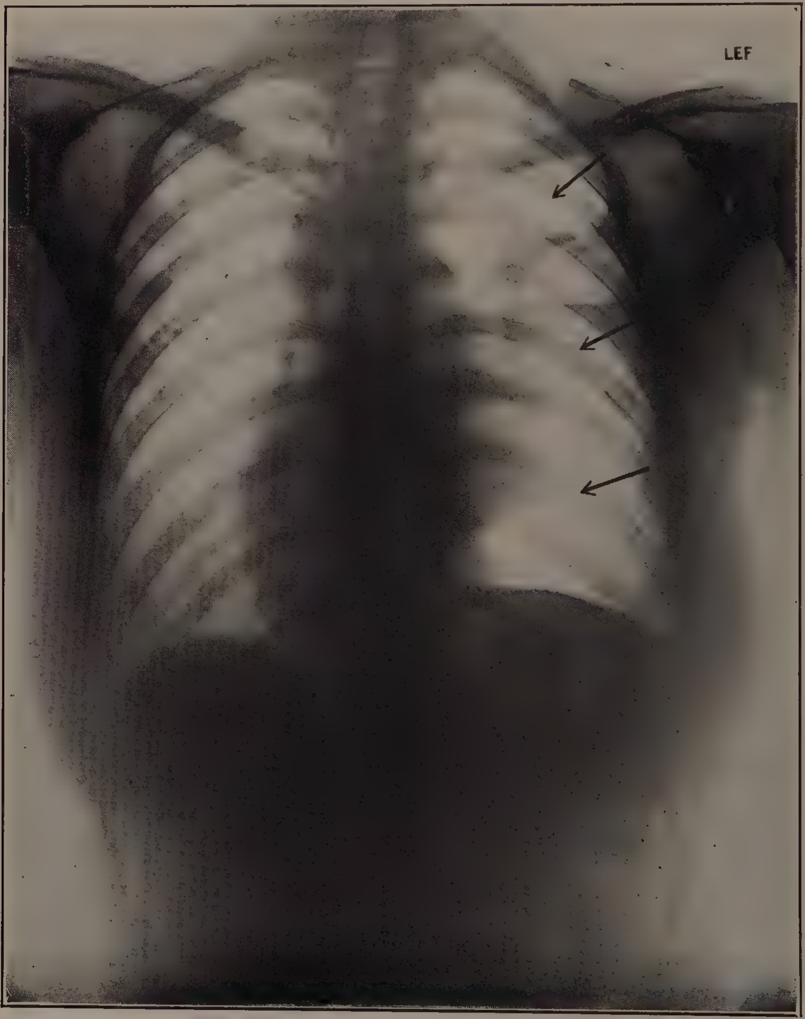

Fig. 295.—Radiograph of hydropneumothorax. Upper arrow indicates area devoid of lung markings. Middle arrow indicates small area of incapsulated fluid. Lower arrow indicates area devoid of lung markings.

The voice sounds, as well as the sounds of the heart, are prone to have a metallic, ringing quality engrafted upon them, as may also râles which are audible over the superior thorax in tuberculous cases. Over the distribution of a coexistent effusion respiratory and vocal sounds are abolished.

Three auscultatory sounds which are considered most pathognomonic of pneumothorax, the falling-drop sound, the succussion splash, and the coin test of Gairdner, are to be elicited in the majority of cases. Similarly, in cases with a communicating bronchial fistula the lung-fistula sound can often be elicited during full inspiration. This sound is most frequently audible over the anterolateral thorax, corresponding to the lower portion of the upper lobe of the lung.

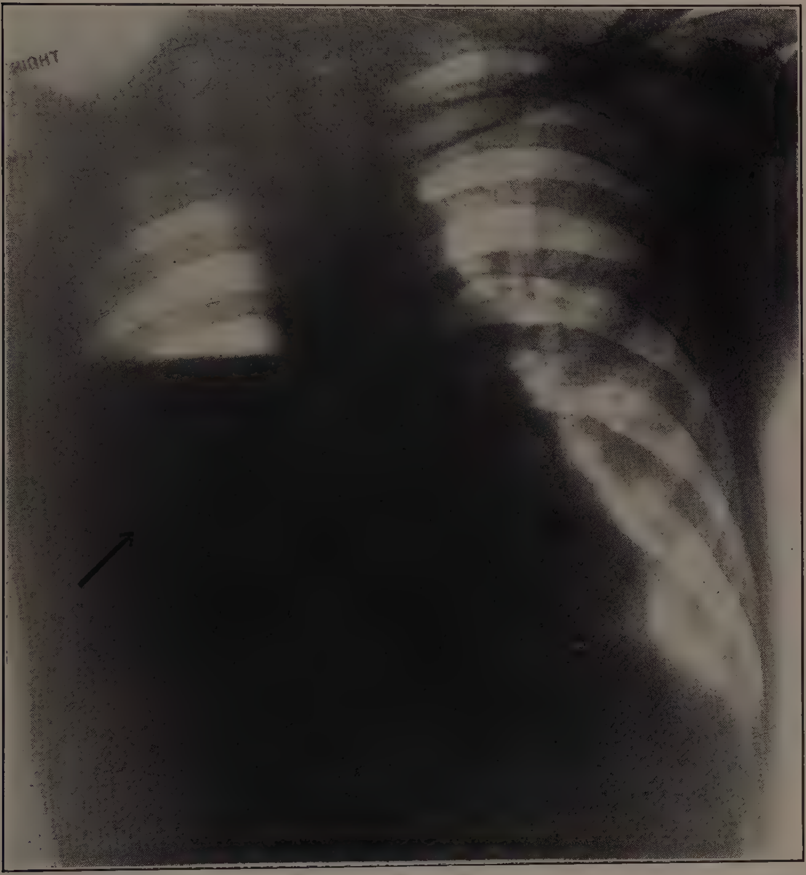

Fig. 296.—Radiograph of hydropneumothorax. Film taken with the subject in the upright position. Note that the upper border of density is horizontal. Arrow indicates density due to fluid.

X-ray of the thorax in the presence of pneumothorax reveals, in the diffuse form of the disease, an extensive area of increased transparency, limited by the chest wall and the border of the compressed lung. In this area bronchial trunk markings are absent, although it may be traversed by shadows referable to pleural adhesions. The diaphragm is depressed on the affected side. Fluoroscopic examination demonstrates the decreased diaphragmatic movement on the affected side, often with exagger-

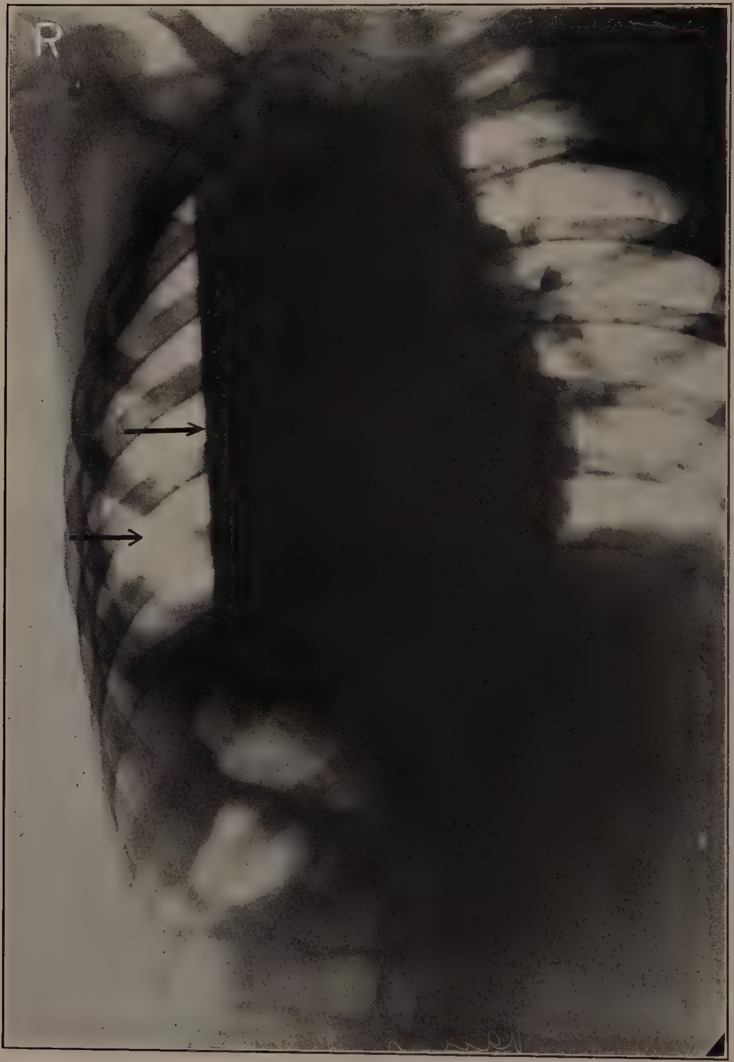

Fig. 297.—Radiograph of hydropneumothorax. Film taken with subject lying on left side. Upper arrow indicates area devoid of lung markings. Lower arrow indicates the horizontal border of density.

ated movement on the normal side. In the presence of effusion the inferior portion of the pleural sac presents a shadow, the density of which varies with the quantity of the fluid. The upper border of this shadow, in the absence of adhesions, is a straight line. The density shifts rapidly with change in position of the patient. Circumscribed pneumothorax is characterized by a limited area of increased transparency, usually localized over the upper lobe, simulating a pulmonary excavation.

Diagnosis.—The physical signs of diffuse pneumothorax present a picture which is so characteristic that an error in diagnosis is infrequent. However, it is possible for confusion to arise in certain cases; and the disease must at times be differentiated from pulmonary excavation by chronic phthisis, and from emphysema, diaphragmatic hernia, hydrothorax, and pleural effusion of moderate degree.

Local or circumscribed pneumothorax involving the superior portion of the pleural cavity may be confused with a *tuberculous cavity* of the upper lobe. In this connection it should be recalled that advanced cases of phthisis are commonly attended by thoracic retraction; that the consolidation of this disease exerts a diametrically opposite influence upon the respiratory and vocal sounds; and that the classical signs of pneumothorax are absent in cases of phthisis, even though the disease is complicated by pleural effusion. Similarly, diffuse pneumothorax may at times be simulated by extensive excavation of a lower lobe of the lung in advanced chronic phthisis. Here, again, the signs attending the two diseases are so distinctive that little confusion should be occasioned.

In the case of *emphysema* careful percussion of the inferior limits of the area of hyperresonance or tympany will reveal in the presence of simple pneumothorax that the resonant area is coextensive inferiorly with the lower limit of the pleural sac. Moreover, the respiratory phenomena attending the two diseases are entirely dissimilar.

Diaphragmatic hernia is commonly attended by signs and symptoms referable to the digestive tract, such as vomiting, colic, or obstipation; radiographic and fluoroscopic studies serve in all cases to clear up the diagnosis.

The differential points between pneumothorax, hydrothorax, and pleural effusion have been discussed under the latter disease.

CHAPTER XL

DISEASES OF THE DIAPHRAGM

The diaphragm is subject to both functional and organic diseases, and is influenced in its action by inflammatory diseases of both the thoracic and abdominal organs. Pressure upon the diaphragm either from fluid, air or tumors above, or from tumors or increased intraabdominal pressure from below, may diminish its excursion.

The functional diseases are usually due to irritation or injury to the phrenic nerve.

SPASM OF THE DIAPHRAGM (HICCOUGH)

Spasm of the diaphragm is due to an irritation of the phrenic nerve, frequently as the result of stomach disorders, and produces hiccoughs. These spasms come at more or less regular intervals, varying from five to twenty-five per minute. They may stop and return without apparent cause. The result of these spasms is a sudden increase of negative pressure in the thorax, accompanied by an inrush of air through the partially closed glottis, producing the characteristic sound of hiccough.

Physical Signs.—*Inspection.*—During the hiccough the lower thorax is seen to suddenly contract and the abdomen protrude. Litten's sign is short and moves very rapidly with each spasm.

Palpation confirms inspection as to the sudden convulsive movement of the lower chest and abdomen.

Percussion and auscultation are of little diagnostic value in hiccough.

The fluoroscope shows a sudden contraction of the diaphragm, often in the midst of inspiratory or expiratory movements.

PARALYSIS OF THE DIAPHRAGM

Clinical Pathology.—Paralysis of the diaphragm may be due to injury or disease of the phrenic nerve or to disease or injury of the central nervous system. One or both sides may be involved, unilateral paralysis being somewhat less common and certainly a less serious condition.

Massive collapse of the lower lobes of one or both lungs follows.

Unilateral Paralysis

Physical Signs.—*Inspection.*—Unilateral paralysis is associated with more rapid respiratory rate, diminished expansion, and absence of Litten's sign on the affected side. Respiratory movements of the abdomen may be lessened but are of normal type. The cardiac apex is displaced toward the affected side.

Palpation confirms inspection.

Percussion may show hyperresonance or dullness over the lower lobe of the lung on the affected side, with cardiac displacement toward the affected side.

Auscultation usually shows bronchovesicular breath sounds and a few moist râles over the base on the affected side.

Fluoroscopic examination shows a high arched immovable diaphragm on the affected side, while on the unaffected side the diaphragm may move with unusual vigor.

Bilateral Paralysis

Physical Signs.—*Inspection.*—Bilateral paralysis is associated with recession of the abdomen during inspiration instead of protrusion. The abdominal viscera do not descend on inspiration. The respirations are shallow and are associated with increased movements of the lower ribs. Litten's sign is absent. The compressive action of the abdominal muscles during defecation and urination are said to be absent.

Palpation confirms the inspection as to the reversal of respiratory movements of the abdomen and the lack of descent of the abdominal organs on inspiration.

Percussion shows hyperresonance or dullness over the collapsed lower lobes. The heart dullness is unchanged.

Auscultation.—Feeble or absent expiratory murmurs over the lower lobes are noted. Usually breath sounds are less intense over the remainder of the lung. Cough and voice sounds are also feeble and a few râles may be heard over the collapsed lower lobes.

X-ray.—The fluoroscopic examination is most useful. It shows the diaphragm high arched with no respiratory contraction. Occasionally a muscular twitching or slight spasm of one or both leaves may be noted. On account of the serious condition of the patient fluoroscopic examination cannot always be made.

DIAPHRAGMATIC HERNIA

Clinical Pathology.—The abdominal contents may protrude into the thorax because of congenital malformation or lack of development of the diaphragm producing true hernia. The hernia may occur through the esophageal foramen, or through the central or lateral portion of one of the diaphragmatic leaves. An injury producing a tear or cut of the muscles may allow abdominal organs to enter the thoracic cavity. This is spoken of as evisceration or traumatic hernia.

The high position of the abdominal organs due to paralysis or relaxation of the diaphragm may at times suggest diaphragmatic hernia.

The symptoms may be slight or severe. Pain is usually of a cramping nature over the lower portion of the sternum. Sometimes an unusual fullness in the chest with cardiac palpitation is noted. Hiccoughs and dyspnea are common. The history of a wound in the region of the diaphragm should suggest careful examination whether symptoms occur or not. Shock is a frequent symptom of the traumatic hernia.

A very high percentage of diaphragmatic hernias are left-sided, due to the protection offered by the liver to the right diaphragm.

Physical Signs.—*Inspection.*—Fullness of the lower left chest with flattening of the interspaces, as in pneumothorax, may be noted if the hernia is very large. Litten's sign is usually absent on the affected side and respiratory movement is restricted.

Palpation may show absence of tactile fremitus and confirms inspection as to the bulging and diminished respiratory movement.

Percussion.—The lower left chest may be tympanic without evident distention in the epigastrium. The cardiac dullness may be masked but is rarely displaced.

Auscultation.—Breath sounds may be absent over the left base near the spine, with gurgling sounds over this area, due to movement of gas in the displaced intestine or stomach.

The x-ray may occasionally show a knuckle of liver protruding through the right diaphragm without other signs and symptoms.

With a left hernia the arch of the diaphragm is usually flattened and the protruding stomach or bowel can be seen above it. This is especially well demonstrated with a barium meal and fluoroscopic examination.

DIAPHRAGMATIC INFLAMMATIONS

The inflammation of the diaphragm is usually secondary to inflammatory disease of the pleura or subdiaphragmatic structures. Trichinae may become imbedded in the diaphragm and produce inflammatory reactions. Pain on respiration over the lower thorax is usually a constant symptom of diaphragmitis.

The physical signs of diaphragmatic inflammation are not easy to distinguish from the diseased process above or below, the extension of which usually produces the diaphragmitis.

Inspection.—Litten's sign is shortened.

Palpable pain is elicited by pressure over the tenth intercostal space to the right or left of the spine.

Percussion is usually of little value.

Auscultation usually reveals disease above the diaphragm sufficient to mask any signs from the diaphragm itself.

The radiograph may show a rather flat diaphragm, and fluoroscopic examination reveals a rather restricted diaphragmatic movement.

SUBDIAPHRAGMATIC ABSCESS

Clinical Pathology.—Subdiaphragmatic abscess may be associated with symptoms of pleural, pulmonary, diaphragmatic, or liver disease, rendering diagnosis at times difficult.

The abscess is usually located on the right side between the liver and the diaphragm, but occasionally occurs beneath the left half of the diaphragm, being bounded below by the left lobe of the liver, stomach, or spleen.

Subdiaphragmatic abscess may result from infection directly transmitted from ruptured gastric or duodenal ulcers, abscess of the liver, or appendicitis, or the infection may be transplanted from some distant focus via the blood stream or lymphatics.

The onset may be sudden, but is usually gradual. The complex picture often presented always contains the elements of sepsis; namely, irregular temperature with leucocytosis. In cases presenting indefinite symptoms centered around the lower thorax or upper abdomen with a septic temperature and leucocytosis, subdiaphragmatic abscess should be considered.

Physical Signs.—*Inspection.*—Litten's shadow may be shortened, indistinct or absent on the affected side. However, if the abscess presents beneath the inner third of the diaphragm, there will be little interference with diaphragmatic movement and Litten's sign will not be affected. The respiratory movements of the abdomen are frequently restricted. If, as occasionally happens, gas production is associated with the abscess formation, the lower thorax may bulge, as with a pneumothorax. In contradistinction to the pneumothorax the bulging is limited by the upper surface of the diaphragm and rarely extends above the sixth rib in the parasternal line.

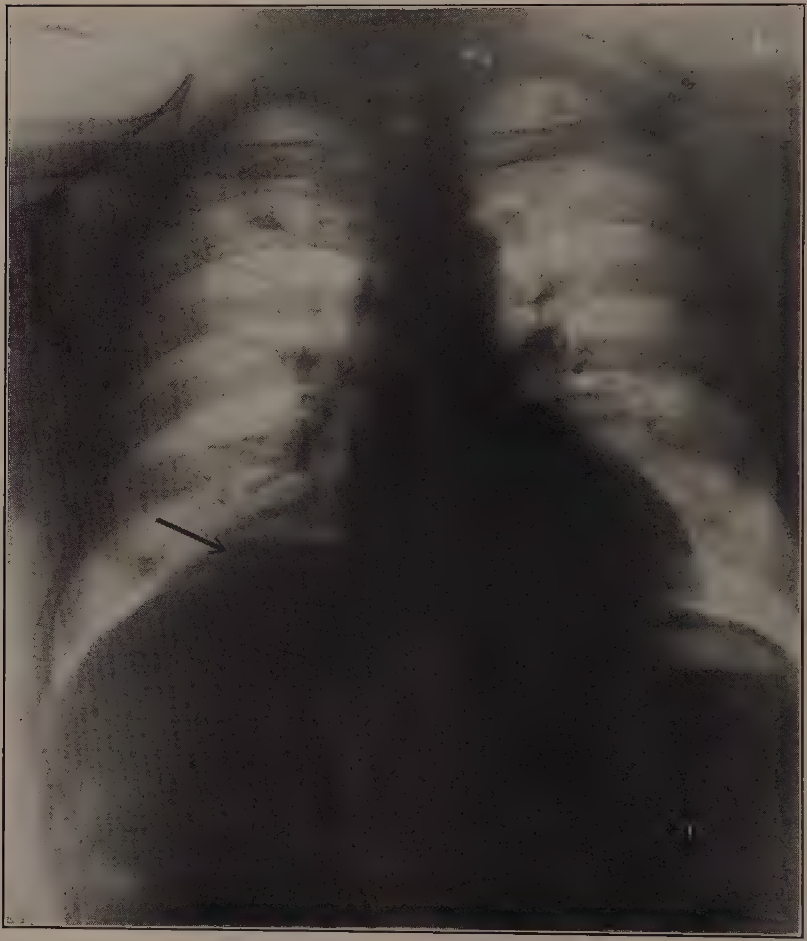

Fig. 298.—Radiograph of subdiaphragmatic abscess. Arrow indicates elevation of the diaphragmatic shadow.

Palpation may reveal tenderness on the affected side over the liver or in the tenth intercostal space near the spine.

Percussion may reveal dullness above the liver, sometimes near the spine and sometimes as a somewhat cone-shaped area near the anterior axillary line or in front. The percussion stroke will often elicit pain over the dull area.

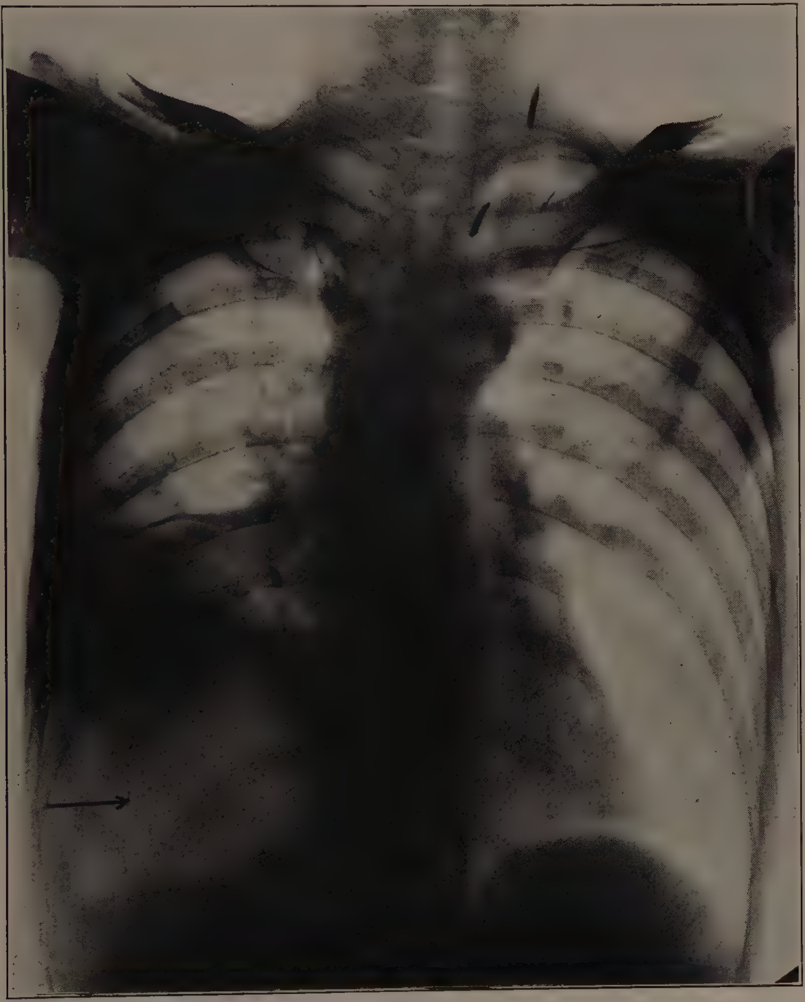

Fig. 299.—Radiograph of subdiaphragmatic abscess. Note elevation of the diaphragmatic shadow. Arrow indicates area of diminished density due to collection of gas between the liver and diaphragm.

If gas is associated with the abscess the dullness will be replaced by tympany. This tympany can be easily determined on the right side, but on the left side it is often difficult to differentiate it from tympany from distended stomach or colon.

Auscultation occasionally reveals a friction rub from rubbing of the diaphragm against the walls of the abscess during respiration. This rub is usually heard in the back, at about the tenth intercostal space, or near the area of dullness.

Should air be present, crepitation may be heard if firm pressure is made with the stethoscope over the lower thorax or liver.

Should the abscess rupture into the pleural cavity or lung, the signs will be those of empyema or lung abscess.

Paracentesis is often a most useful diagnostic procedure. The puncture should be made with a long needle, the site of election being in the tenth intercostal space posteriorly or over the area of dullness.

The x-ray may show localized elevations of the diaphragm, but other symptoms are necessary to determine whether the elevation is due to a tumor or abscess. An area of diminished density below the right diaphragm suggesting air or gas is rarely seen except in abscess. The fluoroscope will demonstrate diminished movement of the diaphragm on the affected side.

Diagnosis depends on a history which may suggest a possible cause for abscess. Signs of sepsis, such as irregular temperature and leucocytosis; shortened, faint or absent Litten's sign; restricted or painful respiratory movement on the affected side, with or without bulging; and tenderness over the liver or in the tenth intercostal space, are suggestive.

Dullness above the ordinary liver dullness, either near the spine and in the axillary line, or in front, and friction rub or crepitation above the liver sometimes aid in making the diagnosis. Exploratory puncture, x-ray and fluoroscopic examination are especially valuable diagnostic procedures when subdiaphragmatic abscess is suspected.

SECTION II

DISEASES OF THE CIRCULATORY SYSTEM

CHAPTER XLI

DISEASES OF THE PERICARDIUM

PERICARDITIS

Inflammation of the pericardium is encountered as a primary and as a secondary disease. The primary cases of the disease occur as a result of trauma to the thorax, and are of infrequent occurrence. The vast majority of cases of pericarditis are secondary to disease in neighboring or remote portions of the bodily economy.

In the secondary form of pericarditis infection reaches the pericardial membrane through the blood stream, the lymphatics, or as a result of direct extension from adjacent structures.

The most prolific sources of such infection comprise the acute infectious fevers, notably rheumatic fever, pneumonia, chorea, scarlatina, pyemia, and cerebrospinal fever. It is estimated that acute rheumatic fever is attended by acute pericarditis in from 6.6 per cent to 24 per cent of cases. In this instance the pericarditis usually develops during the height of the arthritis, but it may precede the acute joint symptoms of the disease.

Lobar pneumonia is attended by pericarditis in approximately 5 per cent of the instances of this disease. The inflammation is prone to assume the purulent form, and it is not infrequently attended by empyema.

Focal infections residing in the tonsils, dental sockets, or other regions occasionally give rise to pericarditis. In this instance the infecting organism is usually the staphylococcus, streptococcus, or pneumococcus.

In certain cases of chronic nephritis, leucemia, scorbutus, and diabetes mellitus pericardial inflammation develops, presumably as a result of diminution of the vital resistance of the host to pyogenic organisms which are harbored in the tissues at various points.

Tuberculosis is occasionally responsible for pericarditis, the inflammation assuming a plastic, serous, or hemorrhagic form. The plastic inflammations are usually acute, whereas the cases attended by effusion tend to pursue a chronic course. The disease probably represents the result of a blood-borne infection, as active pulmonary tuberculosis is usually not in evidence.

Direct extension of disease of adjacent structures, notably of suppurating lymphatic glands, caries of the ribs, and left-sided pleuritis, represents an infrequent cause of pericarditis.

According to the character and distribution of the pathologic changes, pericarditis assumes three principal forms: namely, *acute fibrinous pericarditis; serofibrinous pericarditis,* or *pericarditis with effusion;* and *chronic adhesive pericarditis.*

The pericardium also occasionally harbors fluid of noninflammatory origin, with or without a variable quantity of gas, in the infrequent cases of *hydropericardium, hemopericardium, pyopericardium,* and *pneumopericardium.*

ACUTE FIBRINOUS PERICARDITIS (ACUTE PLASTIC PERICARDITIS; PERICARDITIS SICCA)

Clinical Pathology.—Acute fibrinous pericarditis, acute plastic pericarditis, or pericarditis sicca, represents the initial stage of inflammation of the pericardium. In this form of pericarditis portions of the visceral pericardium originally lose their luster and become clothed with a fibrinous exudate of varying degree. The portion of the membrane first to become involved is usually over the origin of the great vessels, whence the change progresses downward, ultimately involving the parietal pericardium. The distribution of the disease may remain localized to a limited area of the pericardium, or it may be universal, involving a major portion of the two membranes.

The gross appearance of the pericardial surface varies in different periods of the disease. During the early period, in the presence of rather extensive involvement of the membranes, the surfaces present the appearance of two slices of buttered bread which have been apposed and pulled asunder, the "bread and butter" stage of the disease. In other instances the exudate is rolled into irregular folds upon the pericardial surface, constituting the "cor villosum."

In certain instances, instead of involving merely the surface of the pericardium, the inflammation penetrates to the subepicardial muscular tissues, inducing a variable degree of acute myocarditis.

Many cases of acute plastic pericarditis undergo spontaneous resolution without the pouring out of a demonstrable pericardial effusion, but not without permanent damage to the delicate pericardial membrane. In many cases opaque white plaques are left in the membrane, the so-called "milk spots" of this disease. In the presence of more advanced disease, however, with absorption of the exudate adhesions are prone to form between the visceral and the parietal membranes, with the induction of chronic adhesive pericarditis.

The onset of acute fibrinous pericarditis is usually insidious, during the course of one of the diseases which are provocative of the condition; but certain cases are ushered in with a rise or recrudescence of fever, or by pain over the precordia. When pain is present, its character and distribution are fairly characteristic of the disease. In many cases it is represented by a feeling of constriction in the region of the precordia, while in other instances pain is lancinating and is referred along the course of the phrenic nerve. In such cases two tender pressure points are at times to be demonstrated: one situated between the inferior insertions of the sternomastoid muscle, and the other in the costoxyphoid angle between the ensiform cartilage and the insertion of the costal cartilages.

Acute fibrinous pericarditis may terminate favorably within a few days from its onset, if the inflammation does not eventuate in effusion of clinical degree. In the latter event the course is prolonged, and depends in a large measure upon the etiologic factor of the disease.

Physical Signs.—*Inspection.*—In acute fibrinous pericarditis inspection reveals few signs in any event, and it may prove entirely negative. In cases associated with precordial pain, however, the respirations are partially inhibited, and the trunk is apt to be more or less immobilized, as voluntary movement is frequently attended by aggravation of the pain.

Palpation.—Frequently negative in this type of the disease, palpation of the precordia in a spare subject occasionally reveals pericardial friction fremitus over the distribution of the diseased membrane. However, fremitus is more often absent than present, and is not to be expected in the robust subject. Pressure over the points of referred pain may elicit tenderness in cases which are attended by pain.

The pulse is moderately accelerated, but presents no characteristic features.

Percussion reveals no deviation from the normal outline of the heart, as the organ is not enlarged in uncomplicated acute fibrinous pericarditis.

Christian describes an area of dullness near the angle of the scapula in cases of fibrinous pericarditis without fluid. This dullness may be very small or may cover a half of the left back. Many of these cases show no signs of pleurisy with effusion. The cause of this dullness is not clear.

Auscultation of the precordia frequently elicits a pericardial friction sound, the pathognomonic sign of the disease. This sound is confined strictly to the precordia, and is not transmitted thence as are endocardial murmurs. The sound is usually most clearly audible along the left border of the sternum, in the fourth and fifth interspaces. In other cases, again, its point of maximum intensity is situated just below the pulmonary area in the second left intercostal space. In cases of extensive pericardial involvement the sound is clearly audible over the entire precordia.

The quality of the sound varies with the stage and the distribution of the disease. In the incipiency of the inflammation the sound is soft and somewhat analogous to the sound which is provoked by gently stroking a silken fabric. With the full establishment of the exudate, however, the sound is rasping and unmusical, and appears to be in very close proximity to the ear.

The intensity of the sound is modified by the state of the superimposed tissues, being most intense in children and subjects of thin chest wall. The intensity of the sound is commonly accentuated by moderate pressure with the bell of the stethoscope, and it is sometimes partially obliterated by firm pressure with the instrument. Likewise, the sound is usually more intense when the patient bends forward, and it varies in intensity from day to day without apparent cause.

The phases of the friction sound correspond roughly with the sounds of the heart, but not with the same accuracy as do endocardial murmurs. In certain cases the friction sound is prolonged beyond the duration of the first or second sound of the heart, while in others they precede or follow one of these sounds, simulating presystolic or diastolic gallop rhythm.

Diagnosis.—The diagnosis of acute fibrinous pericarditis is readily made when the disease is suspected and the friction sound, the pathognomonic sign of the disease, is detected. But, arising as it so frequently does during the course of an acute infectious disease, it is often masked by the other symptoms of the disease and is not suspected or sought for and hence is frequently overlooked.

Differential Diagnosis.—A mistake in diagnosis may arise in failing to differentiate acute fibrinous pericarditis from organic disease of the aortic valve, or from pleuropericardial friction in pleurisy arising during pneumonia or pulmonary tuberculosis. Organic disease of the aortic valve produces a double murmur, systolic and diastolic in time, with a palpable thrill, which may be easily mistaken for the two phases of the pericardial friction sound. But this double murmur corresponds very closely indeed with the events of the cardiac cycle; the murmurs are transmitted from the precordia in different and definite lines; and the disease produces alterations in the character of the pulse, as for instance the water-hammer pulse in Corrigan's disease, and the small wiry pulse of aortic stenosis. The close circumscription of the maximum intensity of the murmur to the aortic orifice is of aid in differentiation; and, moreover, aortic disease causes hypertrophy of the heart with alteration in the cardiac outline. The pericardial friction sound is more superficial and variable than is the aortic murmur, and is influenced by pressure with the bell of the stethoscope, which does not influence the murmur of organic disease of the aortic valve.

The pleuropericardial friction sound resembles in some respects the pericardial friction sound, but it disappears during suspension of respiration following full inspiration.

SEROFIBRINOUS PERICARDITIS (PERICARDITIS EXUDATIVA; PERICARDITIS WITH EFFUSION)

Clinical Pathology.—Serofibrinous pericarditis, pericarditis exudativa, or pericarditis with effusion, represents the second stage of acute plastic pericarditis, when the latter disease is not early aborted. Beginning as an acute fibrinous inflammation of the visceral and parietal pericardium, with the further evolution of the disease a more or less copious effusion of serous fluid containing fibrinous elements is exuded into the pericardial sac.

In the inception of the effusion the fluid collects in the anterior and superior part of the pericardial sac, but it early descends to the inferior portion of the pericardium, accumulating in the dependent portions of the pericardium. Posteriorly the effusion occupies the space between the heart and the vertebral column, while upon the right side it encroaches upon the cardiohepatic angle of Ebstein, and upon the left side it collects laterally to the cardiac apex. It has been definitely established that a pericardial effusion does not insinuate itself between the anterior surface of the right ventricle and the chest wall, as the apex of the heart is elevated by the effusion and maintains its intimate relation with the parietal pericardium and the anterior wall of the thorax. In numerous instances a single effusion fills the pericardium to a variable degree, but in other cases pericardial adhesions divide the effusion into two or more loculi.

The degree of effusion in serofibrinous pericarditis is variable, usually not exceeding 300 to 400 c.c. More extensive effusions are sometimes noted, however, Corvisart having reported effusions of 1200 c.c. and Gosselin an effusion which attained 2000 c.c.

Purely serofibrinous effusions are colorless or lemon-yellow and contain flocculi of fibrinous exudate, desquamated endothelial cells, and a variable number of red and white blood cells. The specific gravity of the fluid usually approximates 1.018, and it usually contains 4 per cent of albumin, which serves to differentiate it from a simple transudate.

As in the case of acute fibrinous pericarditis, so also in the serofibrinous form of the disease the inflammatory process may invade the subepicardial tissues and induce an acute myocarditis, with impairment of the functional integrity of the heart. Likewise, endocarditis has frequently been found as a coincident disease.

Serofibrinous pericarditis may terminate by resorption of the fluid and a return of the pericardium to a relatively normal state; but such a favorable issue is seldom observed. As a rule, fibrous adhesions are formed between the two membranes, with the induction of varying grades of chronic adhesive pericarditis. Again, in certain cases the effusion develops so rapidly that urgent symptoms arise and the disease may terminate by abrupt or gradual cardiac failure.

The onset of the disease is usually insidious during the course of one of the provocative diseases, or it may arise without assignable cause and long remain latent. In cases of tuberculous origin latency is very common, and the effusion into the pericardium is in some cases associated with coincident pleural effusion. Similar masking of the disease is usually encountered in the cases associated with chronic nephritis.

In the case of pericardial effusion supervening upon an acute plastic pericarditis there are no reliable signs of the change in the pathology, save in certain cases in which the pain of the original disease becomes ameliorated, and in which the tender pressure points disappear. Frequently, however, the change occurs without signs definitely pointing toward it.

In nonlatent cases of serofibrinous effusion the subjective symptoms are variable, and bear a definite relation to the degree and the rate of effusion into the pericardial sac. With progressively developing effusion dyspnea becomes more remarkable, sometimes amounting to orthopnea, and in extreme cases is attended by cyanosis. Dysphagia develops in certain cases and is prone to be accompanied by persistent hiccough and vomiting. In unfavorable cases the patient gradually passes into a state of semicollapse, partially as a result of compression of the heart and great vessels, and partially as a consequence of a coincident acute myocarditis.

The course of serofibrinous pericarditis varies from a few weeks to many months, depending upon the etiologic factor of the disease, the degree of effusion, and the state in which the myocardium is left. Frequently the heart is irritable upon exertion for a prolonged period following a pericardial effusion.

The prognosis of serofibrinous pericarditis depends largely upon the exciting cause of the effusion, the vital resistance of the patient, and the amount of myocardial degeneration which accompanies the disease. Cases arising during rheumatic fever and chorea, when not complicated by endocarditis, usually terminate in recovery. Pericardial effusion developing during chronic nephritis and phthisis, on the other hand, is usually fatal. Unfavorable signs in all cases are orthopnea, marked cyanosis, persistent hiccough, and a progressive fall in systolic blood pressure.

Physical Signs.—*Inspection.*—A large serofibrinous effusion in a young child is sometimes attended by local bulging over the precordia; but this finding is more often absent than present in this class of patients; and in adults with rigid chest walls it is not to be expected. Similarly, in young subjects a large effusion may produce epigastric bulging, *Auenbrugger's sign;* but such a finding is the exception and not the rule.

In a fully established effusion there is frequently to be detected a pulsation to the left of the sternum in the third and fourth interspaces, which is usually attributed to elevation of the cardiac apex by the effusion occupying the dependent portion of the pericardium. Ewart maintains that this pulsation is not due to elevation of the apex of the heart, but that it is due to pulsation of the base of the ventricle which is thrust forward by the effusion. Be the cause of the systolic pulsation what it may, when combined with certain changes in the cardiac dullness, it possesses diagnostic significance. In certain large effusions, on the contrary, for reasons little understood, the cardiac impulse sometimes becomes invisible and impalpable. In such cases when the impulse is not visible in the recumbent posture it may become so when the patient sits up and inclines the trunk forward.

The respirations are moderately accelerated, and in large effusions the dyspnea frequently attains to the degree of orthopnea, with or without a variable degree of cyanosis. The left side of the thorax is apt to expand in a deficient fashion as a result of compression of the left lung by a large effusion. Rarely, indeed, does the examiner encounter tortuosity of the superficial veins of the thorax, arising as a result of increased intrathoracic tension.

Palpation.—Palpation of the precordia is of service in localizing pulsations, and palpation of the base of the neck frequently reveals an exaggeration of the carotid pulse. In spare subjects pericardial friction fremitus is occasionally to be noted early in the course of the disease. When present, it tends to disappear with the development of the effusion, and to reappear with absorption or evacuation of the fluid.

Frequently progressive pressure exerted beneath the left costal arch is provocative of pain in serofibrinous pericarditis.

The *pulse* is commonly moderately accelerated, but not out of proportion to the fever, unless myocardial degeneration has supervened. The rhythm is usually regular in effusions of moderate degree, though it is known that pericarditis is prone to excite extrasystoles, a type of rhythm which is rare in young children. In the presence of massive effusions, on the contrary, the pulse is usually accelerated, of small volume, and of the pulsus paradoxus type, becoming progressively smaller during full inspiration.

The systolic blood pressure varies with the state of the myocardium. A rapidly falling pressure precedes cardiac failure in massive effusions.

Percussion.—With the development of effusion in the pericardium the area of cardiac dullness is naturally extended to a variable degree and in certain directions. In the presence of a massive effusion this area of dullness is irregularly pear-shaped with the base resting upon the diaphragm. During the earlier periods of effusion, when the effusion does not exceed 250 c.c., the earliest demonstrable extension of cardiac dullness occurs toward the left over the base of the heart, where it may reach the second, or even the first intercostal space.

Extension of the dullness to the right of the sternum in the fifth interspace, encroaching upon the normal resonance of Ebstein's cardiohepatic angle, constitutes Rotch's sign of pericarditis with effusion. As Blechmann pointed out, in order to elicit the sign the patient must occupy the sitting posture with the trunk inclined forward.

A further potent finding in pericardial effusion is the extension of dullness below the point in which the cardiac impulse is visible or palpable. The dullness in this instance is caused by the accumulation of the fluid in the dependent portion of the pericardium, in the sinus formed by the arch of the diaphragm and the chest wall below the heart. Dullness in this region, combined with elevation of the visible pulsation of the heart, was regarded by Potain as the most reliable sign of the disease. The only other conditions which may cause similar findings are cardiac hypertrophy and dilation, and under these circumstances the cardiac impulse is not elevated, but is depressed.

Bouillaud demonstrated that when a subject of pericardial effusion passes from the dorsal decubitus to the sitting posture the area of absolute cardiac dullness is extended upward for a distance of two to three centimeters. This change can be demonstrated only in the presence of moderate effusions, and in the absence of coincident effusion into the left pleura.

A coexisting hypertrophic emphysema may serve to obscure the dullness of a fairly large pericardial effusion by the interposition of the voluminous anterior pulmonary borders between the pericardium and the chest wall.

Auscultation.—During the incipient stage of the disease, prior to the development of effusion, a friction sound is sometimes audible over the precordia. With the accumulation of the effusion this friction sound gradually disappears, although frequently persisting at the base of the heart. If inaudible when the patient is in the recumbent posture, it occasionally appears when he is placed in the sitting position.

The cardiac sounds become distant and muffled with the development of the effusion, sometimes as a result of the intervention of the fluid, in other instances as a result of endocarditis or myocarditis. The onset of endocarditis is frequently indicated by a systolic blow, audible over the cardiac apex. As Potain pointed out, the pulmonary second sound is frequently accentuated at the height of the disease.

As a rule, the cardiac action is accelerated, but the rhythm is regular. The detection of extrasystoles in a child presenting symptoms of pericarditis is very suggestive, as this rhythm is not frequently encountered during childhood.

In the presence of a massive pericardial effusion the base of the left lung is compressed and rendered partially atelectatic, with the induction of impaired resonance, bronchovesicular or bronchial breath sounds, and bronchophony over the left base posteriorly, constituting Bamberger's sign of the disease. To elicit the sign it is essential that the patient occupy the sitting posture, with the trunk inclined backward, as the phenomena disappear when the patient leans forward or occupies the knee-chest position.

The radiograph in extensive pericardial effusion shows a flask-shaped shadow over the precordia, with its base directed downward toward the diaphragm and its apex reaching the second, and at times, the first rib. The shadow extends into the right side of the thorax, where it may occupy nearly as much space as it does upon the left side. The left border of the cardiac shadow loses its normal curve, and differentiation between auricle and ventricle is impossible. The left side of the diaphragm may be depressed in extensive effusion. On fluoroscopic examination pulsation of the cardiac chambers is indistinct, and it is not possible to separate auricular and ventricular pulsation. Small effusions, on the other hand, cast no shadows which are pathognomonic of the disease.

The Electrocardiogram.—In some cases of serofibrinous pericarditis it has been observed that the electrocardiogram shows a suggestive high branching of the *T* wave or a shortening of the *R-T* interval somewhat similar to findings occurring in cases of coronary occlusion. The explanation proposed is that the fluid interferes to some extent with the coronary circulation.

Diagnosis.—Precordial pain which is accentuated by pressure over the lower end of the sternum, an initial friction sound which becomes distant or is abolished with the development of fluid, heart sounds which are distant or muffled, a more or less pear-shaped area of precordial dullness, elevation of the cardiac impulse with dullness below the area of pulsation, dyspnea, fever, and an accelerated or irregular pulse, point strongly to effusion into the pericardium.

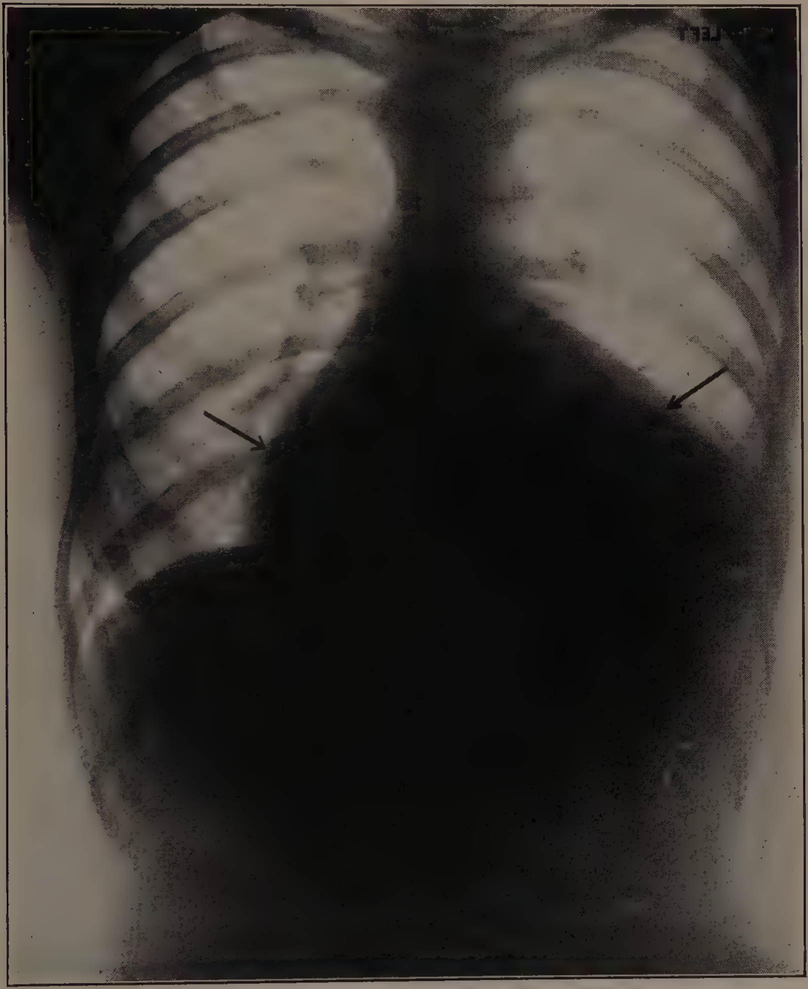

Fig. 300.—Radiograph of pericarditis with effusion. Note flask-shaped increase of cardiac density to right and left of sternum.

The history of an acute infectious fever, nephritis, phthisis, or the detection of focal infection at one or more points further strengthens the diagnosis.

The radiograph of the precordia at the height of the disease establishes the diagnosis, and paracentesis reveals the nature of the effusion.

Differential Diagnosis.—Dilatation of the heart produces increase in the area of the heart, but in this disease there are sufficient differential signs to distinguish the two conditions. In cardiac dilatation the cardiac impulse instead of being obscured is distinctly visible and wavy; the shock of the cardiac valves is distinctly palpable in dilatation; the area of cardiac dullness in dilatation is not pear-shaped, neither does it, except in the case of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces without visible or palpable impulse; rarely indeed is the enlargement of the heart in dilatation so large as to compress the left lung with the production of dullness and bronchovesicular or bronchial breath sounds over the left lung; in dilatation the cardiac sounds are distinctly audible, frequently with a valvular quality engrafted upon them; and the x-ray findings are entirely different in the two diseases.

Serofibrinous pleurisy occasionally must be differentiated from pericarditis with effusion when the effusion reaches an extreme grade. In left-sided pleural effusion, unless the effusion assumes the encysted form, it is likely during a casual examination to be mistaken for a large pericardial effusion; and similarly a large pericardial effusion may be difficult to differentiate from effusion into the left pleural sac. But in pleurisy with effusion the cardiac impulse is displaced; the flatness extends around the side of the thorax; the compressed lung yields Skodaic resonance in the infraclavicular and mammary regions; Traube's similunar space of gastric tympany is obliterated; and the spleen is apt to be displaced downward. In serofibrinous pericarditis, on the contrary, the effusion is apt to extend well to the right of the sternum (Rotch's sign), which is of aid in the differentiation of the two diseases.

CHRONIC ADHESIVE PERICARDITIS (ADHERENT PERICARDIUM)

Clinical Pathology.—Chronic adhesive pericarditis usually represents the terminal stage of acute pericarditis, extensive disease usually developing a number of years after the original acute attack. The disease is especially prone to develop after acute pericarditis complicating rheumatic fever. In other instances chronic adhesive pericarditis develops as an independent disease, usually as a result of infection with the tubercle bacillus.

According to the distribution of the adhesions, four groups of cases are recognized. In the first group only a limited number of adhesions are present between the visceral and the parietal pericardium. In these cases the adhesions are prone to involve the least mobile portions of the heart, such as the interventricular furrow, the border of the left ventricle, the outer side of the right auricle, and the interval surrounding the origin of the great vessels. In the second group of cases the pericardial surfaces are adherent throughout their extent and the pericardial sac is obliterated. In such cases there is seldom much thickening of the membranes, and their structure is little altered. In the third group, in addition to adhesions between the epicardium and the visceral pericardium, there are adhesions between the external surface of the pericardial sac and the adjacent structures, constituting *chronic mediastinopericarditis.* The pericardium may be found more or less intimately bound to the thoracic wall, pleura, diaphragm, or mediastinal structures, and under these circumstances systole of the ventricles is attended by local retractions of diagnostic significance. Similarly, constriction of

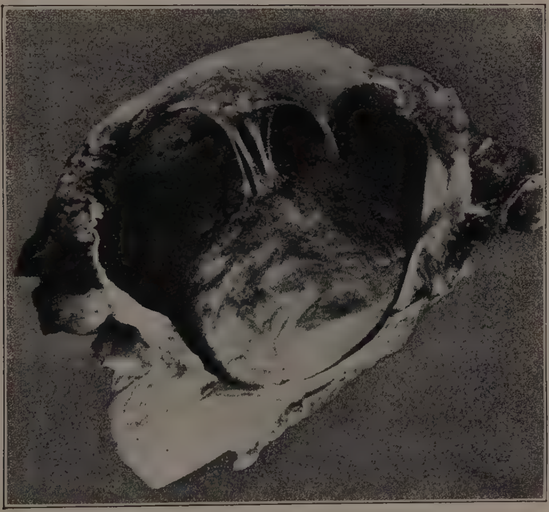

Fig. 301.—Pericardial adhesions. (From Delafield and Prudden.)

the great veins may lead to signs of localizing value. Such cases are often attended by engorgement of the liver, ascites, and edema of the lower extremities. They are sometimes referred to as multiple serositis or Pick's disease. In the fourth group of cases there are adhesions between the external surface of the sac and adjacent structures, but the internal surfaces are free from adhesions. Such cases are rarely encountered.

The condition of the heart varies with the distribution of the adhesions, but the viscus rarely escapes serious damage. In the case of complete obliteration of the pericardial sac the heart is of normal size or is moderately atrophic. This change has been attributed to malnutrition of the myocardium as a result of diminished arterial supply and to actual compression of the heart by the sclerotic pericardium. In the other groups, on the other hand, the heart is increased in size, partially as a result of coincident valvular disease, myocardial degeneration, interstitial myocarditis, or chronic renal disease, and partially as a result of the adherent pericardium constricting the great vessels arising from the heart.

The onset of chronic adhesive pericarditis is so insidious that the disease is seldom detected until extensive adhesions have resulted in serious embarrassment of the cardiac function. Indeed, the presence of a limited number of internal adhesions results in little derangement of the circulation and a few subjective symptoms, hence the disease often remains latent for a long period.

The commonest symptoms comprise shortness of breath upon exertion, and minor degrees of cardiac insufficiency which do not seem to be accounted for by the signs which are to be elicited upon examination. In other instances the disease is manifested largely by digestive disturbances which are occasioned by passive congestion of the abdominal organs.

The prognosis of the disease depends upon the amount of myocardial and valvular damage which attends the pericardial changes. As a rule, in mediastinopericarditis the prognosis is altogether more grave than in the case of internal pericarditis.

Physical Signs.—*Inspection.*—In young subjects, when the disease is attended by considerable cardiac hypertrophy, there is occasionally to be noted moderate bulging of the precordia, and the cardiac impulse is usually displaced from its normal site. In addition to displacement, the apex beat does not exhibit its normal range of lateral mobility upon placing the patient in the lateral decubitus.

External adhesions frequently induce a systolic retraction of the chest wall in the region of the cardiac apex or posteriorly below the left scapula in the tenth and eleventh interspaces, *Broadbent's sign.* In studying such phenomena the patient should suspend the respiration in order to avoid confusion with the respiratory movements of the thorax.

Occasionally, as a result of adhesions between the pericardium and chest wall or left leaf of the diaphragm, the excursion of the left side of the thorax is moderately restricted during inspiration.

Friedreich's sign, diastolic collapse of the jugular veins, is noted in certain cases of adherent pericardium, as is also *Kussmaul's sign,* inspiratory overfullness of the same veins.

The diaphragmatic shadow is sometimes restricted or abolished upon the left side as a result of partial immobilization of the muscle by adhesions.

Palpation.—In chronic adhesive pericarditis there is not infrequently demonstrable a distinct diastolic shock upon palpation of the precordia in the region of the base of the heart. Potain attributed the shock to the elastic recoil of the thoracic wall as it returns to its normal position during diastole. When this shock is encountered in conjunction with systolic retraction of the anterolateral chest wall in the region of the cardiac apex it is very suggestive of adherent pericardium.

The *pulse* is in no wise characteristic of the disease. While the pulsus paradoxus is sometimes in evidence, the radial pulse may be unaltered or may exhibit any of the irregularities which attend myocardial degeneration.

Percussion.—Percussion usually reveals an increase in the transverse dullness of the heart, which may be due to coincident hypertrophy or dilatation, and is in no wise indicative of pericardiac adhesions. More important, is an increase in the area of absolute cardiac dullness at the expense of the area of relative dullness of the heart. Extension of the absolute dullness indicates that external adhesions have bound the heart and pericardium to the anterior wall of the thorax, thus bringing a broader area of the heart into relation with this wall.

A further important sign of chronic adhesive pericarditis is the invariability of cardiac dullness during the two phases of respiration. As a rule, during inspiration the anterior pulmonary borders are interposed between the heart and chest wall and serve to restrict to a variable degree the area of dullness occasioned by the heart. In the presence of pericardial adhesions which prevent the excursion of the pulmonary borders during inspiraton such a restriction of cardiac dullness is not noted, and the cardiac outline is unmodified by the respirations.

Potain stressed immobility of the apical portion of the area of cardiac dullness with change in the patient's posture as an important sign of pericarditis with adhesions. This is true of adhesions involving the apical portion of the pericardium; but in adhesive pericarditis the adhesions may involve the middle region or base of the heart and not interfere at all with the mobility of the cardiac apex.

Auscultation.—Auscultation yields no signs which are characteristic of chronic adhesive pericarditis. A coarse friction sound is sometimes audible, but it is merely indicative of pericardial inflammation, and does not suggest the presence of adhesions. The character of the heart sounds varies with the state of the myocardium. In the presence of coincident cardiac hypertrophy they are accentuated; whereas, if cardiac dilatation has supervened, they are muffled and possess a more or less valvular quality. Similarly, murmurs of coexistent valvular disease are frequently audible, but they are not dependent upon the pericarditis for their production.

The radiograph usually shows a general increase in size of the cardiac shadow. The differentiation between auricles and ventricles is difficult or impossible and frequently the cardiac border is hazy or jagged as a result of adhesions. Rarely calcium deposits may be observed as dense irregular masses imbedded in the thickened pericardium. Fluoroscopic examination shows a diminution of visible pulsation, and restriction of movement during respiration. Likewise, the lateral mobility of the heart is diminished or abolished in cases of extensive external adhesions. Study of the diaphragmatic movements frequently reveals a relative or absolute immobilization of the muscle upon the side of the disease. Cases associated with a limited number of internal adhesions yield no radiographic signs upon which a diagnosis may be made.

The Electrocardiogram.—Since the heart normally changes its position in the chest with change of bodily position, electrocardiograms recorded with the patient lying on one side, supine, and on the other side, normally show differences in the amplitude or in the direction of the initial ventricular complex in the various leads. The electrocardiographic test for adhesive pericarditis consists in making records in all three leads with the patient in each of the above three positions. The absence of comparable differences in electrocardiograms taken in these positions constitutes an important sign of pericardial fixation. It will be observed that there is no particular form of ventricular

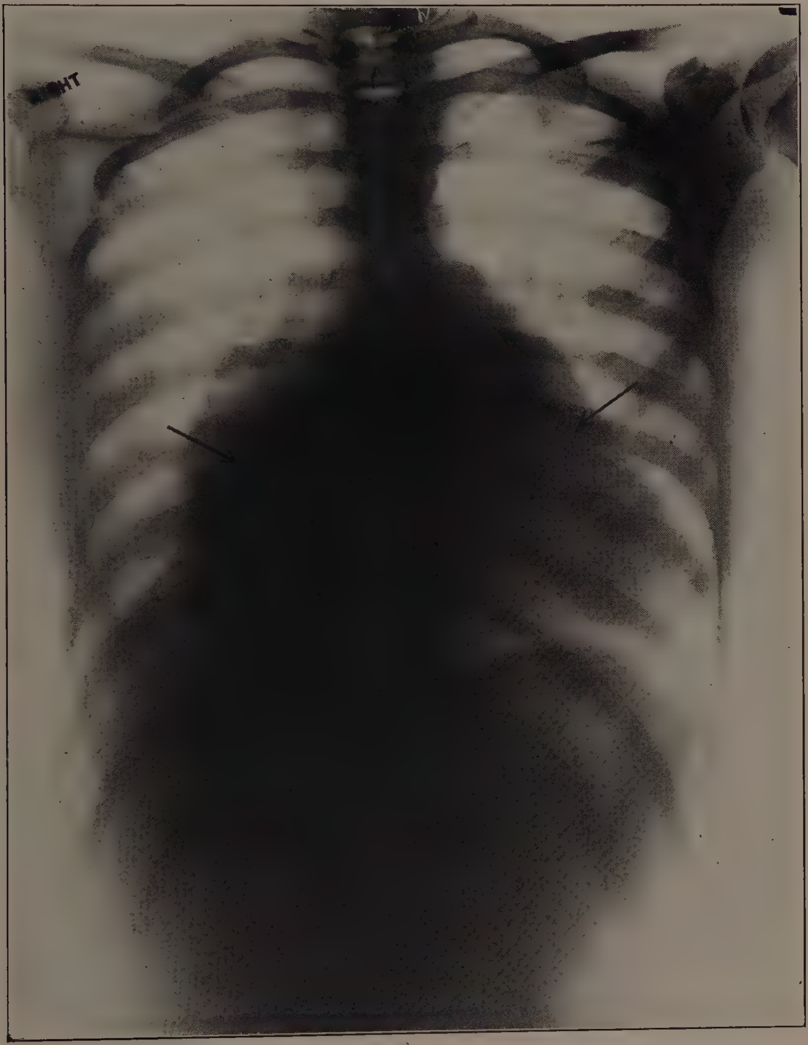

Fig. 302.—Radiograph of pericarditis. Note increase in size and irregularity in shape of heart shadow with greatest density to the right of the sternum, the result of contraction of pericardial adhesions.

complex indicative of pericardial fixation. It is the failure of the initial part of the ventricular complex to show adequate change with change in the patient's position which is significant. Like other valuable signs it alone is not diagnostic but must be considered along with other evidence.

Diagnosis.—In cases in which only a limited number of adhesions exist between the two pericardial membranes, causing little embarrassment of the cardiac action, the diagnosis is attended by great difficulty and is usually impossible.

In cases of more extensive adhesions, involving as well adjacent structures, the diagnosis is to be based upon the localizing signs which have been detailed in the preceding paragraphs. A history of rheumatic fever with complicating pericarditis during previous years is of aid in diagnosis; and when signs of cardiac enlargement and insufficiency are more in evidence than the accompanying valvular disease seems to justify, the examiner should think of the possibility of chronic adhesive pericarditis.

HYDROPERICARDIUM (HYDROPS PERICARDII)

Clinical Pathology.—Hydropericardium, the transudation of serous noninflammatory fluid into the pericardial sac is usually a sequence of general anasarca of chronic nephritis or imperfectly compensated cardiac disease. In rarer instances the serous transudation is a result of thrombosis of the cardiac veins. Again, in certain cases the disease results from compression of the veins by a large aortic aneurysm or mediastinal tumor.

The fluid which occupies the pericardium in this disease is a true transudate, lacking the essential elements of inflammatory fluids. The specific gravity is usually 1.015 or less; the fluid contains approximately 2.5 per cent of albumin; and spontaneous coagulation does not occur. Microscopically, the fluid contains desquamated endothelial cells, but fibrinous elements are absent, as are blood cells in the absence of coincident hemorrhage.

In certain cases the fluid has a semitransparent, milky appearance as a result of admixture with chyle, *chylopericardium.*

The volume of fluid is usually small, and does not produce localizing symptoms or embarrassment of the cardiac action, the clinical picture being that of the provocative disease.

Physical Signs.—Owing to the small amount of fluid which is usually present, the cardinal signs of effusion into the pericardium are rarely in evidence. When localizing signs are present, the disease is differentiated from pericarditis by the absence of fever, precordial pain, and pericardial friction. Naturally, the disease is usually latent and is frequently missed during physical examinations.

X-ray findings are identical with those of serofibrinous pericarditis.

HEMOPERICARDIUM

Clinical Pathology.—The accumulation of blood or sanguineous fluid in the pericardial sac occasionally follows stab wounds or penetrating wounds of the pericardium from other causes. It may be due to rupture of the ascending portion of the aorta, which is enveloped by the pericardium, in which event the disease is rapidly fatal, or it may follow rupture of a coronary artery. Occasionally the fluid of serofibrinous pericarditis is tinged with blood.

Physical Signs.—The physical signs of hemopericardium are those of effusion into the pericardium. Added to these signs in certain cases are signs of internal hemorrhage, as pallor, rapid weak pulse, dyspnea, and collapse. In grave cases death ensues early from pressure upon the heart.

X-ray findings are identical with those of serofibrinous pericarditis.

The presence of blood in the pericardial sac can be demonstrated by use of the aspirating needle.

PYOPERICARDIUM (PURULENT PERICARDITIS; PERICARDIAL ABSCESS)

Clinical Pathology.—Pyopericardium, or purulent pericarditis, is most frequently noted during childhood, occurring as a complication of pneumonia, pyemia, tuberculosis, or an acute infectious fever. The fluid varies in appearance from a thin, semiopaque effusion to thick creamy pus. The predominance of pus cells is in each instance the characteristic feature of the effusion, which also contains fibrinous elements.

The volume of the effusion varies from a few hundred cubic centimeters to a liter or more. Spontaneous absorption of a purulent effusion is exceptional, although the superior strata may be absorbed, leaving a fibrinous mass in the dependent portion of the pericardial sac, which results in the formation of adhesions. Likewise, perforation of the pericardium is not an infrequent complication; and, unless the fluid is evacuated surgically, the case usually terminates in a condition of profound sepsis and ultimate dissolution. Even following surgical evacuation of the fluid, chronic adhesive pericarditis is the usual sequel.

Physical Signs.—The physical manifestations of purulent pericarditis are, in the main, those of effusion into the pericardial sac. However, a friction sound is frequently absent in the early stage of the disease; and if it appears, it is of relatively brief duration. In massive effusions signs of visceral displacement are more remarkable than in serofibrinous effusions.

The aspirating needle is the surest means of diagnosis.

The x-ray findings are identical with those of serofibrinous pericarditis.

PNEUMOPERICARDIUM, HYDRO-, HEMO-, OR PYOPNEUMOPERICARDIUM

Clinical Pathology.—Pneumopericardium, the presence of gas in the pericardial sac, is usually associated with the presence of serous fluid (hydropneumopericardium), blood (hemopneumopericardium), or pus (pyopneumopericardium). The disease is but rarely encountered.

Pneumopericardium may result from penetrating wounds of the pericardium; from perforation of the pericardium by a tuberculous cavity of the lung, gangrene of the lung, or pneumothorax; or it may be spontaneous, owing to the development of the bacillus aerogenes capsulatus of Welch. Malignant disease of the esophagus or stomach very rarely causes perforation of the pericardium and consequent pneumopericardium.

Physical Signs.—The disease is occasionally attended by precordial bulging, with absence of the visible cardiac impulse. A pericardial friction sound is occasionally audible and in other instances succussion fremitus is demonstrable. Percussion of the normal area of cardiac dullness in pneumopericardium yields hyperresonance or tympany, while below the level of the fluid in the pericardium the percussion note is dull or flat.

The heart sounds are feeble, being obscured by the pericardial succussion sound, which is a churning sound that has been compared to the sound produced by a water wheel in motion. Occasionally a pericardial friction sound is audible.

X-ray examination shows the heart surrounded by a halo of diminished density bounded medially by the heart and laterally by the parietal pericardium. If fluid is present the fluoroscope may demonstrate its movement with each cardiac systole.

Diagnosis.—The pericardial succussion sound is pathognomonic of the disease, and its demonstration renders the diagnosis positive. Pneumopericardium is apt to be confused with left-sided pneumothorax or gaseous distention of the stomach. In left pneumothorax the area of cardiac dullness is not obliterated, and the cardiac impulse is usually visible, though frequently displaced toward the opposite side of the thorax. On the other hand, the tympanitic note of a distended stomach disappears when a stomach tube is passed and the gas is liberated from this viscus.

CHAPTER XLII

DISEASES OF THE ENDOCARDIUM AND VALVES

ENDOCARDITIS

Inflammation of the endocardium is most frequently limited to the portion of the membrane which is reflected upon the cardiac valves (valvular endocarditis), although occasionally the portion of the membrane lining the cardiac chambers is also involved (mural endocarditis).

The endocardium is composed of a basal stratum of connective tissue containing a few smooth muscle and elastic fibers, and covered with a single layer of endothelial cells. Subjacent to the endothelial layer is a thin stratum of areolar tissue, containing numerous blood vessels.

The cardiac valves are composed of avascular fibrous connective tissue, over which the endocardial lining of the chambers of the heart is reflected. The valves contain muscular fibers and blood vessels at their attached margins solely, and these vessels do not extend outward into the fibrous portions of the valvular segments.

Inflammation of the endocardium may be occasioned by bacterial invasion, by the toxins of bacteria, or by chemical irritants circulating with the blood. Bacterial agents and toxins are operative during attacks of diphtheria, lobar pneumonia, acute rheumatic fever, tonsillitis, chorea, pyorrhea alveolaris, and acute pyemia. In this connection, it seems very probable that lues and possibly other diseases of spirochetal origin may be operative in the production of endocarditis. In addition to bacterial invasion, endocarditis may result from the irritation of poisoning with lead, phosphorus, alcohol, and nicotine, as well as from the toxins elaborated during diabetes, gout, and other metabolic disturbances.

The organisms which are most frequently obtained upon blood culture of cases of endocarditis comprise the Staphylococcus albus and aureus, ordinary streptococcus, small streptococcus (viridans, tenuans, saprophytic, and modified of Rosenow), pneumococcus, gonococcus, meningococcus, bacillus coli, bacillus influenzæ, and bacillus pyocyaneus.

Endocarditis occurs in three clinical forms; namely, as *acute simple endocarditis, acute infectious endocarditis,* and *subacute infectious endocarditis.*

ACUTE SIMPLE ENDOCARDITIS

Clinical Pathology.—This is the type of endocarditis which is most frequently observed in young subjects. In the vast majority of cases it arises as a result of acute rheumatic fever, and for this reason is sometimes denominated rheumatic endocarditis. While endocarditis may occur in a case of rheumatic fever in which the arthritic symptoms are insignificant, as a rule it occurs in polyarticular cases of the disease in which the general infection with the etiologic agent is extensive. The initial attack of arthritis seems to be most frequently attended by endocarditis, and the younger the subject the more prone is endocarditis to develop.

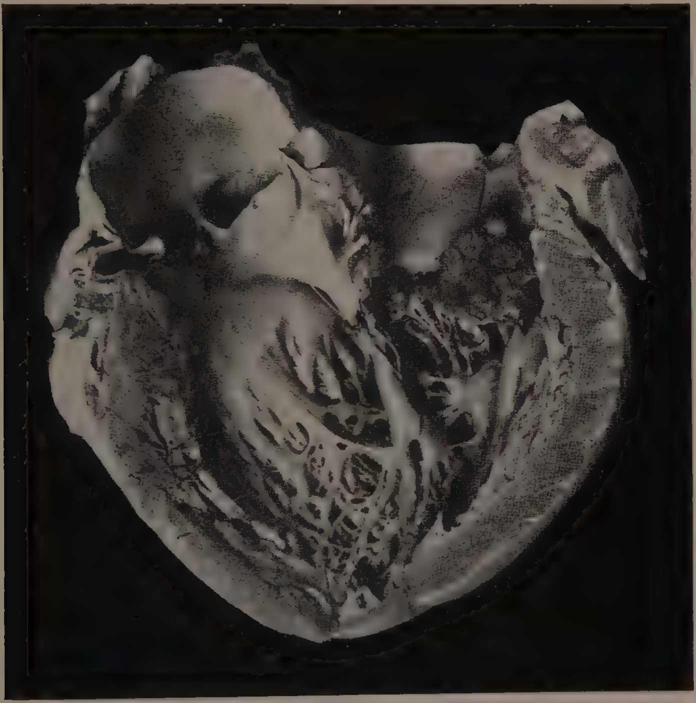

Fig. 303.—Endocarditis, verrucose form. (From Delafield and Prudden.)

Chorea and tonsillitis are likewise prolific causes of this form of endocarditis in young subjects. In other instances the disease follows pneumonia, gonorrhea, focal infections, and terminal infections in subjects of tuberculosis, carcinoma, nephritis and diabetes.

Acute simple endocarditis almost invariably involves the valves upon the left side of the heart, and the mitral valve is involved with greater frequency than is the aortic. In grave cases the chordæ tendineæ may likewise be involved. The pathologic changes occur upon the auricular surface of the mitral valve and upon the ventricular aspect of the aortic valve. The lesions are located along the line of maximum contact of the valvular segments, approximately 2 mm. from the free edges of the cusps.

The initial alteration in the endocardium covering the cusps is a roughening and loss of luster, which develops as a result of necrosis of the endothelial cells. As a consequence of this destruction of the endothelial surface, there occurs a deposition of fibrin in the form of bead-like excrescences or vegetations, measuring usually from 1 to 4 mm. in diameter. Newly formed connective tissue is prone to invade the vegetations and to transform them into areas of fibrosis, which in contracting produce permanent deformity of the valve, with the eventual induction of chronic valvular disease. In late cases calcium salts are deposited in the sclerotic tissues, with the production of more extensive impairment of valvular function.

The myocardium rarely escapes injury during an attack of acute endocarditis. A variable degree of acute myocarditis, which is most marked in the myocardium at the base of the affected valves, commonly attends the endocardial disease. In other cases the myocardial degeneration is more extensive in distribution and plays a greater rôle in impairing the cardiac function than does the valvular disease which is induced. If the conducting system is involved in the retrogressive changes, an altered rhythm is apt to be induced.

Acute simple endocarditis is not attended by symptoms which may be considered pathognomonic of the disease. Indeed, it is frequently difficult to recognize the development of the endocarditis during the course of the original causative disease. A moderate elevation of temperature is commonly in evidence, but it is usually attributed to the original disease rather than to an endocarditis. But when a secondary rise of temperature occurs after a case of rheumatic fever in a young subject, our attention is directed to the endocardium as a probable cause. In a limited number of cases the subject complains of precordial distress and is troubled with repeated attacks of cardiac palpitation, symptoms which usually point to a coincident myocarditis.

Physical Signs.—*Inspection.*—In acute simple endocarditis inspection frequently proves negative throughout the course of the disease, although the onset of the endocarditis may be accompanied by a more pronounced cardiac impulse, and the apex-beat may be displaced toward the left side to a variable degree.

Palpation.—Careful palpation of the precordia sometimes reveals an exaggeration of the valve shock over the apex or the base. Similarly, the cardiac impulse can frequently be determined to be moderately increased in force. It is rare that a palpable thrill is to be detected over the apex or base.

Percussion of the precordia in acute simple endocarditis seldom yields data of diagnostic value. In the subject with an unduly thin chest wall an extension of cardiac dullness to the left and upward may occasionally be elicited at the height of the disease.

Auscultation.—As a rule, the earliest auscultatory sign of simple endocarditis is a muffling and prolongation of the first heart sound at the apex. With the passing of time, this impurity of the sound usually gives place to a systolic murmur with its point of maximum intensity localized over the mitral area, and transmitted for a variable distance toward the left axilla. The transmission of the murmur indicates organic change in the valve, as the murmur which is induced by mere yielding of the mitral ring is not transmitted to any appreciable distance from its point of greatest intensity.

In an initial attack of acute simple endocarditis the murmur is nearly always systolic in time, as the toxin of rheumatic fever seems to have a special predilection for the mitral valve. A presystolic or diastolic murmur points to former disease of the valves and to a condition of chronic valvular disease. The pulmonary second sound is commonly moderately accentuated.

Occasionally the murmur of acute simple endocarditis is to be detected at the aortic area, but the mitral valve bears the brunt of the disease in the vast majority of cases. In isolated instances murmurs are audible over both valves. Also occasionally a systolic murmur is audible over the pulmonary valve, a murmur which is functional in type and which is not produced by endocarditis.

Diagnosis.—While the elicitation of a mitral systolic murmur during the course of an acute infectious fever or in association with chorea or tonsillitis is the most pertinent finding in the diagnosis of acute simple endocarditis, the mere presence of such a murmur does not constitute a conclusive sign of the disease, as a relative murmur is prone to develop at this valve during febrile states, as a result of yielding of the mitral ring. In reaching a diagnosis of endocarditis the physical signs must be construed in conjunction with the previous history of the case, and in certain instances a positive diagnosis must be reserved for some time.

The presence of a diastolic murmur points, as a rule, to former valvular disease, while extension of cardiac dullness, and rapidity and irregularity of the pulse point to myocarditis rather than to endocarditis.

ACUTE INFECTIOUS ENDOCARDITIS (ACUTE SEPTIC ENDOCARDITIS; ULCERATIVE ENDOCARDITIS; MALIGNANT ENDOCARDITIS)

Clinical Pathology.—This form of acute endocarditis is usually encountered in adult subjects, most frequently between the ages of thirty and forty-five years. Children are seldom attacked, and the disease is likewise rarely associated with rheumatic fever or chorea. In the majority of cases it develops as a result of septicemia arising from a septic wound, puerperal sepsis, erysipelas, gonorrhea, or pneumonia. With less constancy the disease develops as a sequel or complication of scarlatina, measles, diphtheria, or influenza. Impairment of vital resistance to infection by alcoholism, overwork, and debilitating disease appears to bear some causal relation to the attacks of the disease.

The organisms which are most frequently recovered from the lesions or cultured from the blood stream comprise the Streptococcus hemolyticus, Staphylococcus aureus and albus, gonococcus, and pneumococcus. Less frequently the meningococcus, diphtheria bacillus, or influenza bacillus is the causative organism of the disease.

Pathologically, two types of acute infectious endocarditis are recognized; namely, the *vegetative* form, and the *ulcerative* form of the disease.

In the *vegetative* type of malignant endocarditis the pathologic changes are very similar to those of the acute simple form of the disease, except that the vegetations attain a more luxuriant growth, occasionally attaining the size of a small cherry. The most extensive lesions are usually encountered in cases of gonococcic and pneumococcic origin. When the vegetations upon the valvular cusps are luxuriant, they frequently cause a moderate stenosis of the valvular orifice. Likewise, as the vegetations are quite friable, small fragments are apt to become detached and to occasion embolism of distant organs or tissues of the body.

In cases of extensive disease the valvular lesions frequently extend to the chordæ tendineæ, and occasionally they involve the endocardium lining the cardiac chambers (mural endocarditis).

In the *ulcerative* type of acute infectious endocarditis the initial change in the valvular endocardium is the formation of one or more patches of yellowish color, and with an irregular surface. In this area desquamation of the endothelium usually occurs. The denuded area is soon invested with a reddish thrombotic mass of variable thickness. In extreme cases of ulceration fenestration of a valvular cusp may occur, as may also abscess or aneurysm of the cardiac wall.

In acute infectious endocarditis the several valves of the heart are involved in the following order of frequency: mitral, aortic, mitral and aortic, tricuspid, and pulmonary. In grave cases the chordæ tendineæ and the mural endocardium may participate in the morbid process.

Embolism in distant organs and tissues of the body is a not infrequent complication of acute infectious endocarditis. The sites of predilection comprise the spleen, kidneys, and brain, although petechiæ frequently arise in the skin, conjunctiva and mucous membranes, apparently as a result of embolism. As the embolus is usually of infectious origin, suppuration or abscess formation frequently is noted.

Owing to the infectious basis of this form of endocarditis, the disease is commonly attended by polymorphonuclear leucocytosis of a variable degree.

The course and symptoms of acute malignant endocarditis are inconstant and variable, with the result that at least three distinct clinical types of the disease are recognized; namely, the *cardiac*, the *pyemic*, and the *typhoid* forms of the disease. Certain clinicians recognize a fourth clinical type, the *meningeal* form of acute infectious endocarditis.

Symptoms which are common to all clinical types are fever, sweats, anemia, loss of weight, and frequently, delirium. The onset of embolism is indicated by certain localizing signs and symptoms. Splenic embolism is indicated by pain under the left costal arch, usually attended by tenderness and enlargement of the spleen. Renal embolism is commonly attended by hematuria and tenderness in the renal region. Cerebral embolism is ushered in by visual changes, aphasia, or motor palsies. Peripheral embolism is attended by petechiæ or subcuticular abscess formation.

The *cardiac* form of acute malignant endocarditis usually develops upon a foundation of chronic valvular disease. The onset of the acute endocarditis is marked by a rise of temperature, associated with an accentuation of the cardiac signs, such as precordial distress, cardiac palpitation, arrhythmia and dyspnea. The disease may subside in a few days, or it may persist for several weeks or months. Recurrent attacks are frequent, but recovery from this form of the disease is not infrequent.

The *pyemic* form of acute infectious endocarditis attends states of general pyemia. As a rule, symptoms which are referable to the endocardial inflammation are masked by the signs and symptoms of septic intoxication which are present. The advent of signs of embolism, the development of endocardial murmurs, and symptoms of cardiac insufficiency point toward involvement of the endocardium and heart muscle.

The *typhoid* form of acute malignant endocarditis, which is usually caused by infection with the gonococcus or pneumococcus, resembles the clinical course of enteric fever so closely that observation and laboratory tests are required to differentiate the two diseases. The appearance of signs of embolism, a complication which is less common in this form than in the other types of the disease, points toward an endocarditic nature of the malady.

Acute infectious endocarditis may run a brief course of a few days, or it may continue over a period of weeks or months with or without periods of remission. The majority of the cases terminate fatally as a result of embolism, meningitis, pericarditis, infarction, cardiac aneurysm, or perforation of the myocardium.

Physical Signs.—*Inspection.*—In this form of endocarditis inspection seldom yields any data relative to the endocardiac disease, save in cases belonging to the cardiac group, in which the endocarditis develops upon the foundation of chronic valvular disease. In this instance the cardiac impulse may be displaced from its normal site, and there may be in evidence signs of moderate or extreme cardiac hypertrophy. In cases associated with extensive myocardial degeneration the impulse is usually diffuse or invisible, and is displaced from its normal site in the fifth interspace.

Palpation of the precordia is usually negative in this disease, as the murmurs which are induced by the endocarditis are rarely attended by palpable thrills. In cases developing upon the basis of chronic valvular disease, however, palpation may reveal alterations in the force and in the site of the cardiac impulse.

The pulse is commonly regular, of small volume, low tension, and may be accelerated beyond the degree of pyrexia.

Blood pressure readings are variable, and are influenced largely by the state of the myocardium and the arterial system.

Percussion is seldom of value in the diagnosis of acute malignant endocarditis, as extension of the cardiac dullness is not always to be elicited. Occasionally in the cardiac form of the disease, however, it is possible to demonstrate an increase in cardiac dullness in one or more directions.

Auscultation affords the most reliable signs of acute infectious endocarditis, and is the method of physical examination chiefly relied upon in reaching a diagnosis of the disease. The abrupt appearance of a systolic murmur at the mitral area during one of the causative diseases of endocarditis is very suggestive of the disease. Moreover, the murmur of acute infectious endocarditis exhibits certain characteristics which are of aid in diagnosis. The murmur is quite variable, and is apt to present changes in quality and intensity upon consecutive examinations. Its sudden appearance during an acute disease or a septic state is likewise suggestive of infective disease of the endocardium. Moreover, the subsequent appearance of a second murmur, notably a diastolic murmur at the aortic area, superimposed upon a systolic mitral murmur, is very suggestive of the progressive involvement of the valves by the disease.

The friction sound of a complicating pericarditis is occasionally to be detected, and myocardial changes may occasion alteration in the intensity and quality of the cardiac sounds.

Diagnosis.—The diagnosis of acute infectious endocarditis rests largely upon the previous history of the illness and upon the auscultatory signs enumerated, arising in a subject of an acute infection or in a chronic septic state. The sudden appearance of the murmur and changes in its intensity and quality upon daily examinations speak strongly in favor of an acute infectious endocarditis.

Just as pyogenic organisms can be cultivated from the lesions in approximately 90 per cent of cases, so also routine culture of the blood during life may occasionally reveal the causative organism of the disease.

Differential Diagnosis.—The febrile movements of *typhoid fever* and *malaria* are differentiated by the specific tests for these diseases.

Acute miliary tuberculosis may occasion confusion for a time; but in the presence of this disease it is usually possible to detect a tuberculous focus at some point; and, moreover, tuberculosis is commonly attended by leucopenia. Petechiæ are usually present in grave cases of endocarditis, and are absent in miliary tuberculosis. Culture of the blood stream and examination of the spinal fluid are the deciding factors in the differentiation of the two diseases in many instances.

SUBACUTE INFECTIOUS ENDOCARDITIS (SUBACUTE MALIGNANT ENDOCARDITIS; SUBACUTE BACTERIAL ENDOCARDITIS; CHRONIC ULCERATIVE ENDOCARDITIS; ENDOCARDITIS LENTA)

Clinical Pathology.—Subacute infectious endocarditis is much more common than is the acute infectious type of the disease. The cases usually develop during adult life upon the basis of chronic valvular disease. The infecting organism is in the vast majority of cases, in 95 per cent of cases according to certain investigators, a nonhemolytic streptococcus (Streptococcus viridans). With less frequency the disease is due to infection with the gonococcus or influenza. The original focus of infection usually resides in a tooth socket, the tonsils, intestines or gall bladder; and, in marked contrast to acute malignant endocarditis, the original causative disease usually causes very little constitutional disturbance.

The valvular changes are limited largely to the left side of the heart, attacking by preference the mitral and aortic valves. Right-sided valvular disease is exceedingly rare. This form of endocarditis, however, exhibits a special predisposition to involve the chordæ tendineæ and the mural endocardium.

The vegetations are not unlike those which attend the acute ulcerative type of the disease, save that they are usually smaller, and tend to assume a yellowish, green, or dark red color. In involvement of the mitral valve the vegetations tend to spread upward along the auricular wall. Likewise, when the aortic valve is diseased, the lesions tend to extend backward along the endocardium lining the left ventricle and to involve the aortic segment of the mitral valve. The vegetations are, as a rule, of smaller dimensions than in the acute type of the disease, particularly in disease of the aortic valve. Cases are noted, however, in which large lesions almost occlude one or more orifices of the heart. Fenestration of valvular segments occasionally is found at autopsy, as is also rupture of the chordæ tendineæ.

Embolism is a frequent accompaniment of the disease, resulting in petechiæ or other signs of localizing value.

The onset of the disease is usually insidious, with chilliness followed by an irregular elevation of temperature, precordial distress, and vague pains in the joints. The duration of the disease varies from a few months to one or two years, during which there are exacerbations and remissions of the symptoms and signs of the endocarditis. The vast majority of the cases terminate fatally as a result of cerebral embolism, secondary anemia, terminal pneumonia or renal complications.

Physical Signs.—The physical manifestations of subacute infectious endocarditis are limited largely to the circulatory system. The murmurs of chronic valvular disease are commonly in evidence, and present no characteristics which suggest the onset of a subacute endocarditis, especially when the disease attacks the mitral valve. In the case of involvement of the aortic valve there are frequently indications of progressive cardiac insufficiency and changes in the quality and intensity of a coincident mitral murmur.

Cutaneous petechiæ are frequently in evidence, and the skin of the hands and feet frequently presents small, painful erythematous nodules, which have been considered pathognomonic of the disease.

Diagnosis.—The diagnosis of the disease rests largely upon the incidence of a persistent rise of temperature in a subject of chronic valvular disease rather than upon the cardiac signs which may be in evidence. The presence of petechiæ and erythematous nodules may be considered corroborative of an endocarditic basis of the affection. Blood culture should be practiced in every suspicious case, as a nonhemolytic streptococcus may occasionally be detected in this manner.

CHRONIC VALVULAR DISEASE (CHRONIC VALVULITIS)

The terms *chronic valvular disease* and *chronic valvulitis* are applicable to a very large group of cases in which progressive pathologic changes in the valvular cusps are demonstrable at autopsy. The myocardium frequently shares in the morbid process as well as the valves, and in certain instances the disease extends to the proximal portion of the aorta or pulmonary artery.

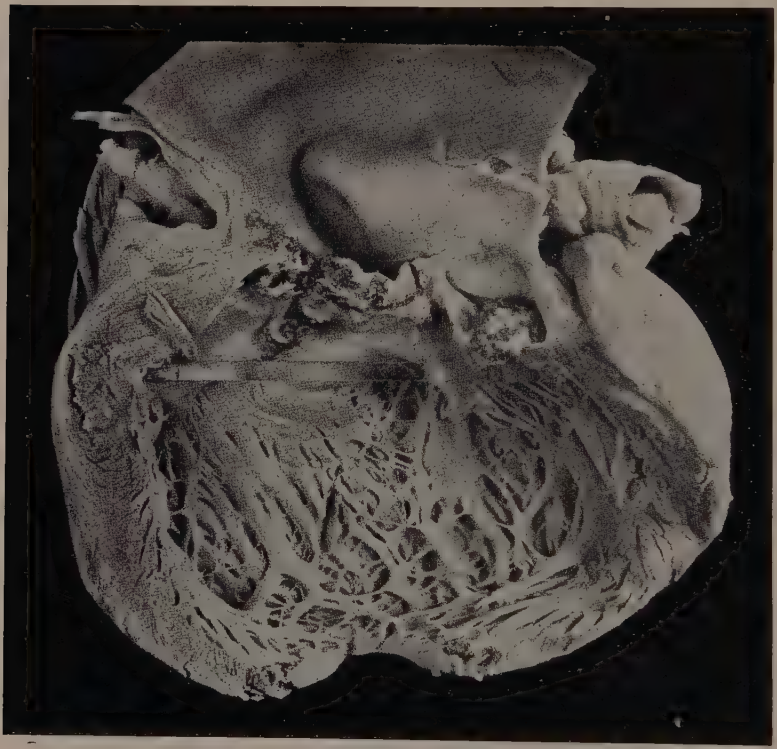

Fig. 304.—Chronic endocarditis. (Delafield and Prudden.)

The etiologic factors which are operative in the production of this large class of cardiacs differ with the age, sex, and economic status of the patients. In children and young adolescents the provocative agent is usually a former acute endocarditis, due to rheumatic fever, tonsillitis, or chorea. In female subjects puerperal sepsis plays an important etiologic rôle. In subjects in middle and advanced life syphilis is an important factor, as are also arteriosclerosis and chronic nephritis. In subjects of laborious occupations, in whom retrogressive changes have occurred in the aortic valve from a former endocarditis, rupture of a segment may attend vigorous effort. Finally, certain of the lesions are congenital, and are in no wise acquired.

The lesions of chronic valvulitis are of two types: *stenotic,* which result in narrowing and partial occlusion of an orifice of the heart, offering an obstacle to the onward flow of the blood stream; and *regurgitant,* which are characterized by the inability of the valvular cusps to prevent the regurgitation of the blood stream during systole. In the production of regurgitation the pathologic change may consist of deformity of the cusps, shortening of the chordæ tendineæ, or temporary relaxation of the muscular ring at the base of the segments, with the result that normal cusps are incapable of closing the abnormally large orifice (relative regurgitation).

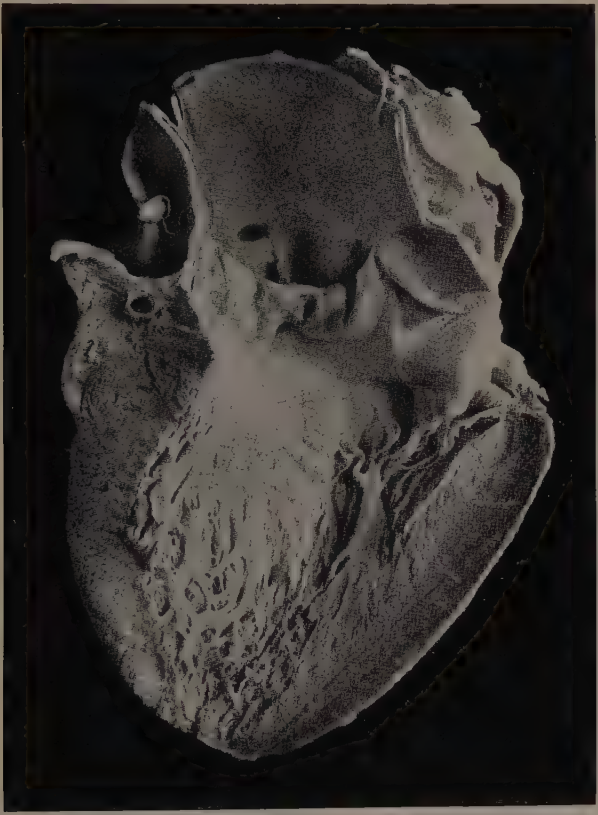

Fig. 305.—Chronic endocarditis with coalescence of two aortic cusps. (From Delafield and Prudden.)

The lesions are largely confined to the mitral and aortic valves, although occasionally involving the valves of the right side of the heart. However, the vast majority of chronic lesions of the valves of the right heart are congenital in origin. Relative regurgitation at one or more of the valves of the right heart likewise occurs frequently as a late result of organic lesions of the mitral and aortic valves.

The valvular lesions may be single or multiple; and the heart presents a combination of organic and relative regurgitation in numerous instances. As a general rule, luetic lesions involve the aortic valve, whereas the residual lesions of endocarditis are more frequently encountered at the mitral valve.

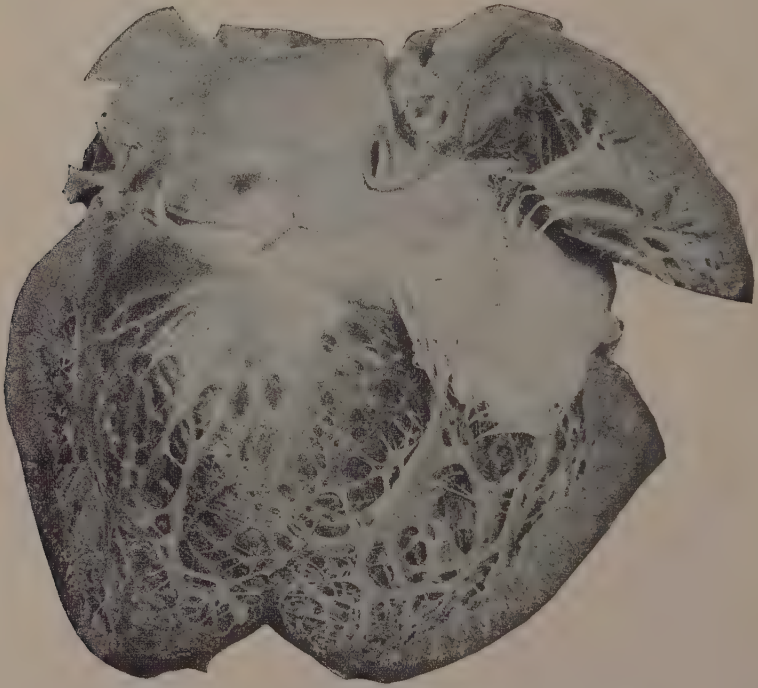

Fig. 306.—Aortic incompetence with hypertrophy and dilation of left ventricle, the result of arteriosclerosis affecting the aortic valves. Note how the valves have been curled, thickened, and shortened, the edges of valves being a half inch below the upper points of attachment. The anterior coronary artery is shown, the lumen narrowed. (Reduced one-half.) (From Warfield.)

Pathologic Physiology of Valvular Disease.—The mechanical effects of valvular lesions are classified as *primary,* as they are exerted upon the heart, and as *secondary,* as they are expended upon more remote organs and tissues of the body.

The natural tendency of a valvular lesion is to diminish the quantity of circulating blood in advance of the lesion, and to increase the quantity of blood in the circulatory system behind the site of the lesion; hence, to lower arterial tension and to raise venous pressure. In the presence of a stenotic lesion the chamber discharging blood through the narrowed orifice can only discharge a portion of its contents during systole, while during diastole the chamber receives a certain physiologic quota of blood from the source which discharges into it. Similarly, in the case of a regurgitant lesion, at each systole the incompetent valve permits a certain portion of the contents of the chamber in advance of the lesion to regurgitate into the chamber behind it, which coincidentally is receiving its physiologic quota from its source of supply. In this manner an imbalance is induced in the circulation, the tendency of which is to produce a diminution of arterial tension and a corresponding increase in venous pressure.

If there were no means of balancing the disproportion between the pressures in the arterial and venous circulations, death would supervene early upon the establishment of a valvular lesion of the heart. But to offset the difference in pressure in the two systems, the chamber of the heart upon which the increased burden is thrown compensates for the incompetence of the valve by hypertrophy of the myocardium, and the imbalance of the circulation is corrected for a variable time. As long as this compensation is maintained, the valvular lesion is unattended by subjective symptoms; but when the reserve power of the myocardium is exhausted and the heart fails to adequately expel its contents, compensation is said to be broken; and signs of cardiac insufficiency rapidly supervene.

The effects of valvular lesions are well illustrated by the sequence of events following regurgitation at the aortic valve. When this valve is incompetent a variable quantity of the blood which is expelled into the aorta during ventricular systole regurgitates into the left ventricle during diastole. An increased burden is then thrown upon this chamber of the heart, which is at the same time receiving its quota of blood from the left auricle during ventricular diastole; and the left ventricle undergoes a transient dilatation, the physiologic "active or compensatory dilatation" of Krehl.

In response to the increased demand for work, however, the left ventricle hypertrophies, and the imbalance between the arterial and venous circulations is restored. As long as this hypertrophy is maintained, no change is noted in the general circulation; but the time ultimately arrives in organic disease of the valve when the ventricular musculature is no longer able to sustain the extra burden, and permanent dilatation of the left ventricle, the ''relaxation dilatation'' of Sahli, supervenes.

The dilatation of the left ventricle is attended by stretching of the muscular ring at the base of the mitral valve, and the normal cusps of this valve are unable to adequately close the orifice, with the result that a variable quantity of blood regurgitates into the left auricle during ventricular systole, the period of the cardiac cycle during which the auricle is receiving blood from the lungs. Under the influence of this double supply and increased load, the left auricle undergoes a transient dilatation, to ultimately hypertrophy in compensation for the valvular insufficiency.

After a variable period this compensatory hypertrophy of the auricle yields to dilatation whereupon, by virtue of the regurgitation of blood from the left ventricle, which the dilated auricle is unable to expel during systole, the blood pressure in the pulmonary circulation is raised, as evinced by accentuation of the pulmonary second sound.

The continuous engorgement of the pulmonary circulation predisposes to the development of catarrhal inflammation of the bronchi, and, in more extreme grades, to edema of the lung or hydrothorax.

The heightened tension in the pulmonary circuit is attended by coincident hypertrophy or transient dilatation and subsequent hypertrophy of the right ventricle, compensating for a time the insufficiency of the left heart. But the right ventricle finally yields to dilatation, with the induction of relative tricuspid regurgitation and engorgement of the right auricle.

The right auricle, which is not capable of any great degree of compensatory hypertrophy, early suffers dilatation, attended by general venous stasis with systolic pulsation of the liver and effusion of fluid into the serous cavities and cellular tissues of the body.

The clinical pathology and physical signs of chronic valvular disease are covered in the several sections dealing with the individual diseases.

AORTIC REGURGITATION (AORTIC INSUFFICIENCY; AORTIC INCOMPETENCE; CORRIGAN'S DISEASE)

Clinical Pathology.—Accurate and complete closure of the aortic valve is dependent upon accurate coaptation of the free borders of the cusps of the semilunar valve, which are forced together by the arterial pressure in the root of the aorta just subsequent to the completion of ventricular systole. When, as a sequel of recent endocarditis, vegetations are formed upon the valvular cusps, accurate coaptation of the valve segments does not occur during ventricular diastole and a portion of the blood which is expelled from the ventricle during its systole is allowed to regurgitate into the ventricular chamber during diastole. Similarly, shrinking and sclerosis of the segments occurring as a result of chronic endocarditis prevents accurate coaptation of the cusps with the induction of regurgitation at the aortic orifice. In this class of cases there are frequently adhesions between the individual cusps. In yet other instances there is fenestration of one or more cusps, arising as a congenital defect, or as the result of ulcerative endocarditis. But there is another group of cases in which, without any disease or deformity of the valvular cusps, the fibrous ring at the aortic orifice is enlarged with the result that the normal valvular cusps are no longer capable of closing the abnormally large aortic orifice, resulting in the induction of *relative aortic regurgitation.* This change is encountered in connection with syphilitic aortitis and aneurysm of the ascending portion of the aorta. On account of the firm fibrinous character of the aortic ring enlargement is less common than with the mitral. In yet another group of cases aortic regurgitation develops as the consequence of a slow sclerosis of the valvular segments and aortic ring, occurring as part and parcel of generalized arterial sclerosis in middle-aged male subjects who have for years followed strenuous occupations. Mineral intoxication, particularly plumbism, is the initiating cause in certain cases, whereas other cases develop in subjects of a gouty diathesis. Aortic regurgitation is prone to develop during middle life, although isolated cases are picked up in younger subjects; and the vast majority of the cases occur in the male sex.

The mechanical influences of the lesion in aortic regurgitation are classified as *primary,* as they are exerted upon the myocardium, and as *secondary,* as they are expended upon more remote organs and tissues of the body. As a result of the progressive and consecutive manifestation of these influences, three distinct stages or periods of the disease may be diagnosed and employed by the examiner as a basis for his prognosis and treatment of the disease.

During the first stage of the disease the brunt of the lesion falls upon the left side of the heart and the arterial system, and the myocardial changes are limited to the left auricle and left ventricle. During the second stage of the disease, with the gradual failure of the left heart, the pressure rises in the pulmonary circulation and the lesion is compensated by hypertrophy of the right ventricle. Finally, the third and terminal stage of the disease is ushered in with failure of right ventricular hypertrophy and engorgement of the general venous circulation.

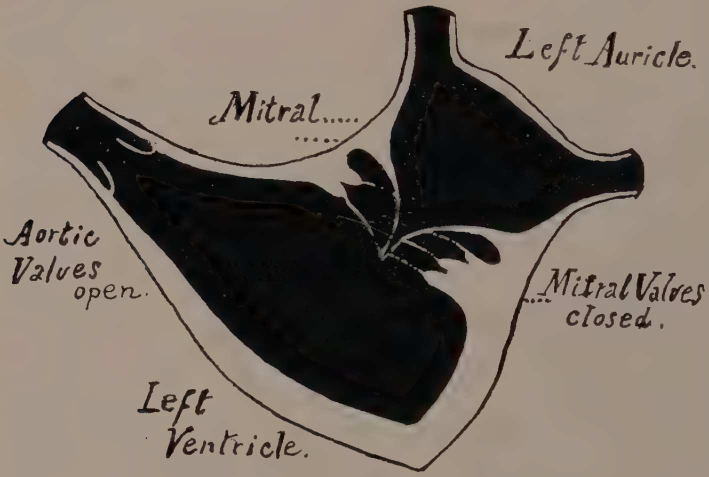

Fig. 307.—Normal ventricular systole. Mitral valve is closed; aortic valve is open.

During the *first stage* of the disease the primary effect of the aortic lesion is exerted upon the left ventricle during its diastole, at which period it becomes the target for two streams of blood entering its chamber simultaneously, the one propelled into the ventricle by left auricular systole, the other regurgitating into the ventricle from the aorta by reason of the incompetent semilunar valves. Under the stress of this added burden the left ventricle undergoes a transient dilatation which is compensated by hypertrophy of the myocardium of this chamber of the heart. So long as this compensatory hypertrophy is maintained, the ventricle is capable of expelling its contents during ventricular systole and no added burden is thrown upon the pulmonary circulation.

Upon the general arterial circulation, however, the regurgitant lesion exercises a decided influence. At each ventricular systole an excessive quantity of blood is expelled into the aorta, and this quota of blood is expelled with undue force by the hypertrophied ventricle. In this manner the arterial system is abruptly surcharged with blood during ventricular systole, resulting in an abrupt rise in arterial pressure, which is almost immediately fol-

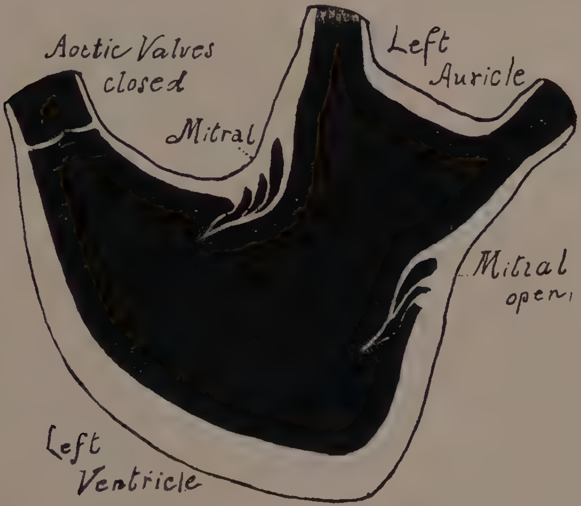
Aortic Valves closed
Left Auricle
Mitral
Mitral open
Left Ventricle

Fig. 308.—Normal ventricular diastole. Mitral valve is open. Aortic valve is closed.

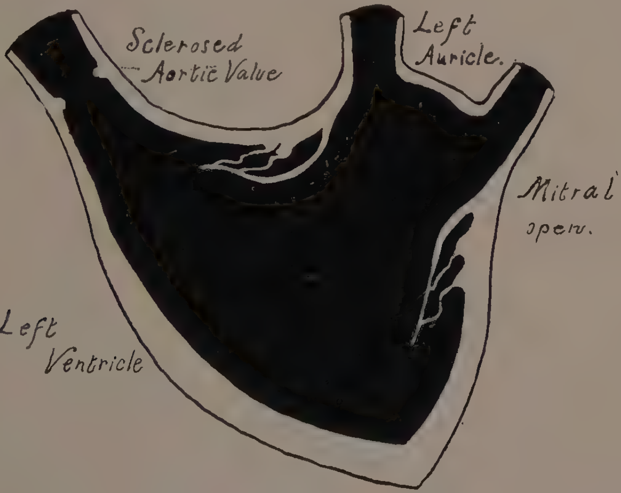
Sclerosed Aortic Valve
Left Auricle.
Mitral open.
Left Ventricle

Fig. 309.—Aortic regurgitation. Mitral and aortic valves are open during diastole.

lowed by as abrupt a fall during ventricular diastole. The effect of the sudden expulsion of the ventricular contents is to distend the arteries which undergo a transient dilatation, with subsequent collapse, producing a pulse wave which not infrequently extends into the capillaries as the capillary pulse of Quincke, and which is occasionally demonstrable in the superficial veins as the centripetal venous pulse. The strain which is thus thrown upon the aorta and general arterial system tends to provoke generalized arterial sclerosis in which the coronary arteries share, with consequent myocardial degeneration.

As the disease progresses, the left ventricle hypertrophies to an excessive degree in its effort to supply the tissues of the body with their normal quota of arterial blood, resulting in the *cor bovinum* of this disease. During the first stage of the disease the subject of the malady is troubled with recurrent transient failures of compensation which are compensated by subsequent restoration of compensation by the left ventricle under repose. Fortunately for the subject, aortic regurgitation is a slowly progressive disease, allowing the left ventricle to compensate by hypertrophy as the regurgitation tends to decompensate by becoming more pronounced. The time eventually arrives when through myocardial weakness the ventricle undergoes dilatation beyond the power of compensation and the mitral valve yields with the establishment of relative mitral regurgitation. In other instances the mitral valve is attacked by endocarditic changes occurring as a result of the continued hypertension in the left ventricle, but the usual mechanism of the establishment of mitral insufficiency is through dilatation of the ventricle.

With the establishment of mitral regurgitation the *second stage* of the disease is ushered in with embarrassment of the pulmonary circulation and compensation by hypertrophy of the right ventricle, as evinced by accentuation of the second sound of the heart at the pulmonic area. In the vast majority of cases the establishment of relative mitral insufficiency during the course of aortic regurgitation is to be considered a distinctly benign complication of the disease, and one which is compensated by right ventricular hypertrophy over a prolonged period of time. In most cases in which relative mitral insufficiency develops, it occurs as a signal that the compensatory hypertrophy of the left ventricle is unable to keep pace with the tendency toward dilatation from nutritional changes in the myocardium, and may be likened to the safety-valve leak of tricuspid insufficiency which for a time relieves the strain thrown upon a laboring right heart later in the course of the disease.

During the second stage of the disease bronchial inflammation is frequently an annoying feature of the case, the intensity of the symptoms and signs varying with the reserve power of the right ventricle.

Compensation by right ventricular hypertrophy is maintained for a variable period in aortic regurgitation; but with the continued hypertension existing in the pulmonary circulation the reserve power of the right heart is overtaxed, and with the supervention of right ventricular failure the third stage of the disease ensues. The right ventricle under these circumstances fails through one of two mechanisms. The tricuspid valve, in the presence of the progressive increase in intraventricular pressure may yield and permit a portion of the contents of the ventricle to regurgitate into the right auricle during ventricular systole in the absence of any definite yielding of the right ventricular wall, with the establishment of the so-called ''safety-valve leak'' of the tricuspid orifice. If, on the contrary, the tricuspid valve retains its integrity in the presence of the progressive rise in the intraventricular pressure, the point is attained when the right ventricle is no longer capable of surmounting the excessive pressure in the pulmonary artery, and the ventricle is only partially emptied during ventricular systole. To the residual blood in the right ventricle is added the contents of the right auricle at the succeeding auricular systole, and the walls of the surcharged right ventricle yield, with dilatation of the ventricle and permanent enlargement of the auriculoventricular orifice. Thus is induced the terminal primary effect of the regurgitant aortic lesion, with the establishment of incompetence of the tricuspid valve with dilatation of the right ventricle.

During this late stage of the disease the secondary effects of the aortic lesion develop in rapid succession. The venous return from the trunk and extremities, pouring into the right auricle from the venæ cavæ, encounters a distinct resistence in the regurgitant lesion at the tricuspid valve. The ultimate result is a marked diminution of the volume of blood which reaches the arterial circulation and a dangerous increase of the volume of blood contained in the venous system. As a diminished volume of blood is expelled at each systole of the right ventricle, the left heart is only partially filled, the left ventricle contracts upon a markedly diminished quota of blood and there is a progressive fall in arterial tension. On the other hand, as a result of the progressive stasis in the venous system, passive congestion of the lungs and abdominal viscera develops, together with effusion into the pleura and peritoneum, and the extravasation of fluid into the cellular tissues of the body.

Aortic regurgitation is not infrequently associated with stenosis of the valve, particularly in cases of endocarditic origin. In these cases the lesion which prevents accurate coaptation of the valvular cusps during ventricular diastole likewise produces narrowing of the orifice during ventricular systole.

In the first and second stages of the disease the subject of aortic regurgitation does not present many symptoms unless he is subjected to sudden strain. There is moderate dyspnea upon exertion, and occasionally considerable nervous irritability. With the failure of compensation by the right ventricle, bronchial symptoms come to the fore and the subject complains of digestive disturbances, frequently of considerable duration. Dizziness frequently occurs from cerebral anemia, and, indeed, sudden death may result from embolism of the brain. Coronary disease may induce attacks of angina, and as the disease progresses dyspnea gives place to orthopnea, with excessive cough and the raising of blood-tinged expectoration.

Physical Signs.—*Inspection.*—In the first stage of aortic regurgitation the cardiac impulse is forceful; its area is more extensive than in the case of the impulse of the normal heart; and the impulse is displaced toward the left and downward as a result of left ventricular hypertrophy. The impulse is observed to be heaving and slightly prolonged, and frequently the area of impulse is surrounded by a circumscribed area of systolic retraction of the thorax. As left ventricular hypertrophy progresses, the apex beat is observed to occupy a site farther and farther from the median line of the thorax. A very characteristic sign of the disease is systolic pulsation visible over the carotids in the lower cervical region, and frequently there are visible pulsations of the subclavian and the brachial arteries. The capillary pulse of Quincke is demonstrable in many cases of the disease and in a smaller number, a centripetal venous pulse is to be detected.

In the second stage of aortic regurgitation there is a striking diminution in the intensity of the signs emanating from the hypertrophied left ventricle as hypertrophy gives place to dilatation. The cardiac impulse becomes less forceful and more diffuse and undulatory, while at the same time being carried farther from the median line and downward. The systolic pulsations of the arteries decrease in force and eventually disappear. The respiratory movements of the thorax now become more frequent and the facies is anxious. Systolic pulsation of the epigastrium is commonly encountered from right ventricular hypertrophy.

During the third stage of the disease the cardiac impulse is very diffuse, feeble, and undulatory, occupying a broad area along the left sternal border between the third and sixth interspaces. The positive venous pulse appears in the jugulars with the establishment of tricuspid insufficiency, and this same state is frequently attended by a systolic pulsation of the liver. The subject suffers with dyspnea and harassing cough, and is no longer able to assume the dorsal decubitus.

Palpation.—In the first stage of the disease with the maintenance of left ventricular hypertrophy the cardiac impulse is heaving and forceful, with great lifting power, and is frequently immediately followed by a moderate retraction of the thoracic wall in the vicinity of the impulse. As left ventricular dilatation becomes imminent the apex beat becomes appreciably enfeebled and rather slapping than thrusting as in the first instance. A diastolic thrill is occasionally to be detected at the aortic area, though a thrill is not present with aortic regurgitation with the same constancy as in the case of stenosis of this orifice. Pulsation of the liver, due to sudden filling and emptying of the hepatic arteries, is often felt.

In the second stage of the disease, with the establishment of mitral regurgitation and hypertrophy of the right ventricle, the cardiac impulse is perceptible farther toward the median line and downward toward the epigastrium. A distinct systolic pulsation of the upper epigastrium is almost constantly present with hypertrophy of the right ventricle.

With the progressive failure of the right ventricle which ushers in the third stage of aortic regurgitation, the cardiac impulse becomes feeble, diffuse, and undulatory, at the same time extending beyond the right sternal margin and well downward into the epigastrium. The positive venous pulse is demonstrable in the jugular vein by compression of the vessel above the clavicle, and systolic pulsation of the liver can usually be detected by bimanual palpation of the hepatic region. This pulsation is of entirely different origin from that noted in the first stage. In the former it is due to sudden filling and emptying of the arteries and is analogous to the visible capillary pulse, while the latter is due to overdistention of veins from back pressure exerted through the venae cavae and hepatic veins.

The *pulse* of aortic regurgitation is of the water-hammer or Corrigan type, with an abrupt distention of the artery which is followed immediately by collapse of the vessel. This character of pulse is readily detected by grasping the forearm just above the wrist and elevating the subject's arm above the level of the heart, whereupon the force of gravity facilitates the emptying of the artery during ventricular diastole. With the inception of mitral regurgitation in the second stage of the disease the volume of the radial pulse is diminished, and with the failure of the right ventricle the pulse is feeble, rapid and running.

Blood pressure readings in aortic regurgitation usually exhibit an increased or normal systolic pressure combined with a very low diastolic pressure, resulting in a very large pulse pressure.

Percussion.—Percussion of the anterior surface of the thorax in aortic regurgitation gives an outline of cardiac dullness which varies directly with the changes which occur in the size and shape

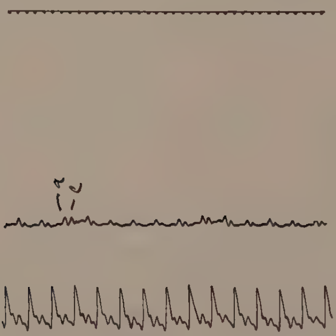

Fig. 310.—Pulsus celer of aortic regurgitation.

of the chambers of the heart. During the first stage of the disease the transverse dullness of the heart is extended to the left and downward as a result of left ventricular hypertrophy. At this period of the disease the apical area is quite discrete and pointed, to become rounded and obtuse with the establishment of right ventricular hypertrophy in the second stage of the disease. At this time the transverse dullness of the heart is increased toward the right of the sternum, where it encroaches upon the vesicular resonance of Ebstein's cardiohepatic angle, and is extended downward into the upper epigastrium. With the supervention of dilatation of the right ventricle the cardiac outline extends further to the right of the sternum and downward into the upper portion of the epigastrium. In cases which are associated with aneurysm of the ascending aorta, percussion of the area of vascular dullness at the base of the heart yields an extension of dullness to the right of the sternum in this region.

Percussion of the pulmonary bases during the first and second periods of the disease yields little, if any, alteration in the character of the percussion sounds elicited. In the third stage of the disease, however, with the establishment of pulmonary edema, there is dullness over the bases posteriorly; and, in the presence of hydrothorax, there is frank flatness over the distribution of the effusion. Similarly, during the terminal stage of the disease the areas of hepatic and splenic dullness are found to be extended, as a result of chronic passive congestion of these organs.

Auscultation.—In aortic regurgitation there is generated a murmur with its point of maximum intensity at the aortic valve area near the junction of the second right costal cartilage with the sternum, over the fourth intercostal space to the left of the sternum, over the middle of the gladiolus, or near the apex of the heart. The point of maximum intensity of this murmur is thus variable; but in the majority of instances it will be localized in the aortic area in the second right interspace. The line of transmission of the murmur passes in the majority of cases downward along the right sternal margin toward the ensiform cartilage, though occasionally it crosses the median portion of the sternum and is conducted to the apex of the heart. The murmur usually replaces the second sound of the heart at the aortic area, but in certain cases both murmur and second sound are audible.

The quality of the murmur is variable during the different stages of the disease, and its quality and intensity are also dependent upon the character of the underlying lesion and upon the degree of regurgitation. During the first stage of the disease with the maintenance of left ventricular hypertrophy with its excessive output of blood at each ventricular systole the murmur is commonly loud and blowing, but seldom as harsh as is the murmur of aortic stenosis. The narrower the orifice through which the blood stream regurgitates, the harsher is the murmur. Similarly a prolongation of the murmur points to a narrow orifice at the incompetent valve. A brief "whiff" at this area, on the contrary, in which the murmur occupies only the early portion of diastole and is quickly attenuated and disappears, points to a wide orifice with the regurgitation of a more considerable quantity of blood.

With dilatation of the left ventricle and the establishment of relative mitral regurgitation during the second stage of the disease the murmur at the aortic area loses much of its intensity, and there is added a soft, blowing systolic murmur at the mitral area, which is transmitted downward and toward the left axilla, and is attended by accentuation of the pulmonic second sound. There is occasionally generated at the mitral area the murmur of Austin Flint, the mechanism of which has been described in a previous section.

With the supervention of right ventricular failure in the third stage of the disease, the murmurs at the aortic and mitral areas become feeble or are entirely inaudible; the formerly accentuated pulmonic second sound gives place to an enfeeblement of this sound; and the systolic murmur of tricuspid regurgitation becomes audible in the tricuspid area.

In every case of suspected aortic regurgitation the examiner should endeavor to pick up a transmitted diastolic murmur over the carotid arteries. During the first stage of the disease this murmur is usually quite distinctly audible, to become enfeebled or lost during the second and third stages of the disease. Similarly, auscultation of the femoral artery will occasionally elicit the double murmur of Duroziez in aortic regurgitation. This sign is usually accentuated by pressure with the stethoscope sufficiently to produce partial occlusion of the artery.

As aortic regurgitation and stenosis not infrequently coexist, the examiner will occasionally detect a double "see-saw murmur" at the aortic area, the one systolic and harsh in quality, the other diastolic and more musical and less intense.

Diagnosis.—The diagnosis of aortic regurgitation is as a rule readily made upon the physical findings which are presented. The presence of a diastolic murmur with its point of maximum intensely localized in the aortic area and transmitted downward and toward the left, the signs of immense hypertrophy or dilatation of the left ventricle, pulsations in the carotid arteries, the water-hammer pulse, and Duroziez's double femoral murmur, are the cardinal signs upon which the diagnosis is based.

The capillary pulse is demonstrable in the majority of cases, and the characteristic blood pressure readings of the disease are of aid in diagnosis.

AORTIC STENOSIS

Clinical Pathology.—Simple stenosis of the aortic valve is the least frequently encountered of all acquired valvular lesions of the heart, and the cases occur more frequently as part and parcel of general arteriosclerosis in middle-aged subjects than as a sequel of acute endocarditis attacking the valvular segments. Thus, in the majority of instances, the valvular change consists of a gradual and progressive sclerosis of the cusps and sclerotic contraction of the aortic ring, resulting in diminution in the caliber of the aortic orifice. Aortic stenosis occasionally arises as a result of congenital malformation of the valve, in which event the lesion may consist of a fusion of the cusps with a mere slit or chink between the free borders of the leaflets. In yet other cases the valve segments are the site of numerous excrescences which project into the lumen of the orifice during ventricular systole with the induction of narrowing of the physiologic orifice of the valve at this period of the cardiac cycle.

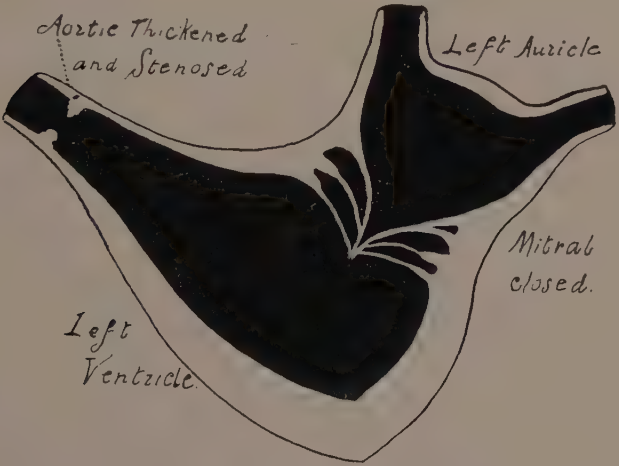

Fig. 311.—Aortic stenosis. Mitral valve is closed during ventricular systole. Aortic orifice is stenotic.

But if organic stenosis of the aortic valve is a rare lesion, there are, nevertheless, a number of possible factors which are capable of generating a systolic murmur at this valve in the absence of organic disease of the aortic ring or segments. In the presence of simple endocarditis or arteriosclerosis the segments may bear minute excrescences which, while producing no actual stenosis of the orifice, are sufficient to produce fluid veins with the generation of a systolic murmur with its point of maximum intensity at the aortic area. Similarly, dilatation of the proximal segment of the aorta just distal to the semilunar valve is capable of generating a murmur at this valve area in the absence of any diminution in caliber of the aortic orifice, constituting in this instance *relative aortic stenosis.* Also in the anemic and debilitated subject, with impoverishment of the circulating blood, functional murmurs which are systolic in time are occasionally generated at the aortic valve. Hence, when a systolic murmur is encountered at this valvular area, the burden is upon the examiner to exclude organic disease of the aortic valve.

As in the case of aortic regurgitation, the course of the disease in aortic stenosis is insidious in onset and extended in its evolution and course, and in its clinical manifestations and its effects upon the myocardium and bodily economy may advantageously be divided into three stages. During the first stage of the disease the myocardial changes are limited to the left side of the heart; during the second stage the burden falls upon the pulmonary circulation and the right ventricle; and during the third stage the mechanical influences of the lesion are expended upon the general venous system with the supervention of right ventricular failure.

As in the case of other valvular lesions of the left heart, the mechanical influences of the aortic lesion are classified as *primary,* as they are expended upon the myocardium; and *secondary,* as they are expended upon the venous system and their influence is felt by more remote organs and tissues of the body.

During the *first stage* of aortic stenosis there is a progressive hypertrophy of the left ventricle in response to the increased demand which is made upon this chamber of the heart in adequately expelling its contents through the narrowed aortic orifice into the aorta. As the onset of the disease is slow and insidious, this hypertrophy develops slowly, keeping pace with the progress of the lesion or with the progressive rise in peripheral resistance in arteriosclerotic cases, with a primary and transient dilatation of the chamber of the ventricle. During this period of the disease compensation is frequently broken by excessive physical exertion upon the part of the patient, only to be restored by repose and freedom from exertion. But with the progress of the disease, the point is reached when the ventricle is no longer capable of compensating for the lesion by hypertrophy, and ventricular dilatation supervenes with yielding of the left auriculoventricular ring and the establishment of relative mitral regurgitation. The establishment of the mitral insufficiency relieves the laboring ventricle by a reduction of intraventricular pressure, but it serves to increase the tension of the pulmonary circulation and to throw an added burden upon the right ventricle, which hypertrophies in response to the increased demand for work.

The *second stage* of the disease, which is ushered in with the induction of relative mitral insufficiency is readily recognized by the accentuation of the pulmonary second sound which ensues upon right ventricular hypertrophy and the increasing hypertension in the pulmonary circulation. The course of the second stage of aortic stenosis pursues the same course as has been detailed in the section upon aortic regurgitation.

The *third stage* of aortic stenosis is ushered in with failure of the hypertrophied right ventricle, the changes occurring in a similar manner and with similar results as in the case of aortic regurgitation.

As a rule, the lesion which produces stenosis of the aortic valve also renders the valve segments incapable of completely closing the orifice during ventricular diastole, so that aortic stenosis and regurgitation frequently coexist in the same subject.

Physical Signs.—*Inspection.*—During the first stage of the disease, with the maintenance of adequate compensation by left ventricular hypertrophy a moderate displacement of the apex-beat outward and downward is frequently the only visible evidence of disease of the cardiovascular system. Upon more minute study the impulse is frequently observed to be increased in extent and to be slow and heaving. There are many elderly subjects of the disease, however, in whom the intervention of the anterior borders of emphysematous lungs between the heart and the chest wall entirely obscures the visible cardiac impulse.

During the second stage of aortic stenosis, with failure of the left ventricle, the cardiac impulse is carried farther toward the left axilla and downward, and is occasionally represented by a broad impulse along the left sternal border in the third, fourth, and fifth interspaces. At this time also, as a result of right ventricular hypertrophy, systolic pulsation of the epigastrium is frequently in evidence. In children and in female subjects precordial bulging is occasionally encountered.

With failure of the right heart in the third stage of the disease, venous engorgement becomes a prominent part of the clinical picture, with frequently systolic pulsation of the jugulars as a sequence of tricuspid leakage, and prominence of the abdomen and swelling of the feet and ankles from the effusion of fluid.

Palpation.—During the first stage of aortic stenosis the findings upon palpation of the precordia are characteristic of the disease. The cardiac impulse is slow, forceful, and sustained, possessing a great degree of lifting power. In the presence of immense left ventricular hypertrophy there is frequently a concomitant negative recoil of the surface adjacent to the impulse. In the latter portion of the first stage, when left ventricular dilatation becomes imminent, there is a progressive diminution in the lifting power of the impulse, which becomes more brief and is suggestive of a slapping movement of the cardiac apex.

Aortic stenosis is attended by a systolic thrill at the aortic area, a thrill the intensity of which is dependent upon the degree of stenosis and upon the contractile power of the left ventricle. The more rigid and unyielding is the aortic ring from sclerosis or calcareous deposit, and the greater the hypertrophy of the left ventricle, the more intense is the systolic thrill at the aortic area. With the advent of imminent failure of the left heart there is a progressive diminution in the intensity of the thrill, which finally becomes imperceptible.

When the left ventricle dilates and mitral regurgitation is established during the second stage of the disease the cardiac impulse loses its forceful lifting character, to become feeble, slapping, and undulatory. The valve shock over the pulmonic area becomes accentuated as a result of right ventricular hypertrophy, and palpation of the hepatic region may reveal a pseudopulsation which is due to the transmitted impact against this organ of the right ventricle during its forcible systole.

During the third stage of the disease the cardiac impulse is feeble and undulatory, with irregularity of both force and rhythm. A thrill at the aortic area is not always encountered at this late stage of the disease.

The pulse in aortic stenosis is slow and sustained for an appreciable interval beneath the palpating finger. Its volume is diminished, the diminution varying with the degree of stenosis and the compensation by left ventricular hypertrophy. With the finger upon the radial artery the examiner is instinctively reminded of the slow and forcible systole of the hypertrophied left ventricle, expelling its contents through a narrowed aortic orifice. The sphygmographic tracing of the pulse of aortic stenosis exhibits a prolonged and oblique anacrotic limb, capped by a prolonged plateau, and followed by a slow descent to the base line. During the last stage of the disease the pulse is rapid, diminished in force and volume, and sometimes disordered in rhythm.

Percussion.—During the first stage of the disease, so long as left ventricular hypertrophy is moderate, little or no alteration may be encountered in the area of cardiac dullness. With the progress of the disease there is a progressive extension of the dullness toward the left axillary line and downward. In the interpretation of percussion findings in this disease the examiner should make due allowance for alveolar distention of emphysema if such is present.

With left ventricular dilatation in the second period of the disease the cardiac outline extends farther to the left and downward, while the region of the cardiac apex becomes rounded or obtuse from the presence of hypertrophy of the right ventricle.

With right ventricular failure in the terminal stage of the disease the cardiac outline extends well to the right of the sternum, encroaching in this situation to a variable degree upon the angle of vesicular resonance of Ebstein at the junction of the areas of cardiac and hepatic dullness.

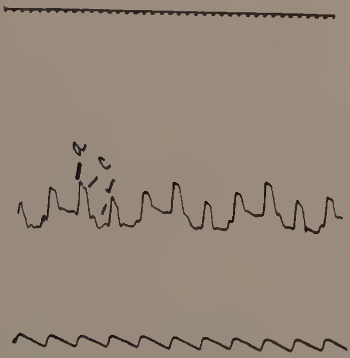

Fig. 312.—Sustained pulsus tardus of aortic stenosis.

Percussion of the bases of the lungs at this late stage of the disease usually yields bilateral dullness arising from pulmonary edema, and occasionally the frank flatness of a hydrothorax is elicited upon one side. Similarly, the areas of hepatic and splenic dullness may be found extended, as a result of enlargement of these organs from chronic passive congestion.

Auscultation.—Aortic stenosis is attended by the generation of a systolic murmur with its point of maximum intensity at the aortic valve area in the second intercostal space adjacent to the right sternal border, whence the murmur is transmitted upward into the great vessels of the neck. The intensity and quality of the murmur are dependent upon the degree of stenosis and power of the ventricular systole. During the first stage of simple aortic stenosis only the one murmur is audible upon auscultation of the precordia.

In the presence of slight endocarditic lesions upon the cusps of the aortic valve, in the group of cases which are included under the term "aortic roughening," the murmur is of minor intensity, is blowing and not unmusical and consumes only the early portion of ventricular systole. Such a murmur is followed by a perceptible interval, after which the second sound is audible with great purity at the aortic area. A murmur of this type is only transmitted a short distance from its point of maximum intensity, and frequently is unattended by a palpable thrill. But in the presence of marked stenosis of the aortic orifice with rigidity of the ring and cusps the murmur is harsh and intense and is transmitted well into the root of the neck. This murmur consumes the whole of ventricular systole, and as ventricular systole under these circumstances is prolonged and sustained, the murmur possesses a commensurate duration, entirely replacing the first sound of the heart at the aortic area, and frequently it is followed by an impure second sound or by the murmur of aortic regurgitation.

During the second stage of the disease the murmur at the aortic area becomes progressively diminished in intensity; its range of diffusion is more closely restricted to the aortic area; and auscultation of the cardiac apex will reveal the blowing systolic murmur of relative mitral insufficiency. At the same time the pulmonary second sound at the second left intercostal space is markedly accentuated as a consequence of right ventricular hypertrophy.

With failure of the right heart during the third stage of the disease there is a progressive diminution of the intensity of the aortic systolic murmur, which frequently becomes inaudible late in the disease. At the same time the pulmonary second sound loses its accentuation with the development of the soft blowing safety-valve leak at the tricuspid orifice.

Auscultation of the bases of the lungs now reveals the presence of numerous moist râles, or the respiratory murmur may be abolished over an extensive area in the presence of effusion into the pleural cavity.

Diagnosis.—The cardinal signs which point toward aortic stenosis comprise a systolic murmur with its point of maximum intensity in the aortic area and transmitted thence into the root of the neck, a feeble second heart sound at the aortic area, a palpable thrill over the site of the murmur, a slow pulse of the pulsus tardus type, and symptoms referable to cerebral anemia, notably vertigo.

While systolic murmurs at the aortic area are frequently encountered, aortic stenosis is seldom detected in the autopsy room. The vast majority of such murmurs are due to pathology extraneous to the aortic valve.

The murmur of *relative aortic stenosis,* which is caused by dilatation of the aorta distal to the valve, is systolic in time, but it is unattended by alterations of the radial pulse or ventricular hypertrophy; and it is attended by pulsations of the carotid vessels, accentuation of the aortic second sound, and an extension of dullness to the right of the sternum.

Functional murmurs are only occasionally ecountered at the aortic area. When present, they are musical, transient, and not transmitted beyond the limits of the precordia. They do not cause alterations in the cardiac outline or the pulse, and are usually encountered in young or anemic subjects.

MITRAL REGURGITATION (MITRAL INSUFFICIENCY; MITRAL INCOMPETENCE)

Clinical Pathology.—Complete closure of the mitral orifice is dependent upon accurate coaptation of the segments of the valve, active traction upon the cusps exerted by the papillary muscles through the medium of the chordæ tendineæ, and a proper degree of contraction of the left ventricle, the three factors interacting in a characteristic manner and in a definite temporal sequence. Accurate coaptation of the free borders of the valvular cusps at the commencement of ventricular systole completely closes the auriculoventricular orifice; the simultaneous contraction of the papillary muscles through the medium of the chordæ tendineæ, by exerting traction upon the valvular leaflets, prevents eversion of the segments into the auricle; while the contraction of the left ventricle diminishes by one-half the caliber of the left auriculoventricular orifice and at the same time converts the circular orifice into an oval aperture, which is effectually closed by the cusps of the mitral valve, supported by the muscular ring at their bases and controlled by the papillary muscles and chordæ tendineæ.

A disturbance in this delicately regulated mechanism may operate through any one of its component parts to produce faulty closure of the valve segments with resulting incompetence. Acute endocarditis by the production of excrescences upon the valvular segments or through ulceration and partial destruction of a cusp;

chronic endocarditis through thickening, sclerosis or adhesion of the cusps; chronic interstitial myocarditis through fibrosis of the auriculoventricular ring which supports the cusps; and sclerosis of the papillary muscles through the exertion of undue traction upon the cusps; all may result in regurgitation at the mitral valve. Similarly, in the presence of left ventricular dilatation and myocardial degeneration, the contraction of the ventricle may prove insufficient to reduce an auriculoventricular orifice which is unduly large, and the chordæ tendineæ may not permit the accurate coaptation of the normal cusps, with the consequent induction of *relative mitral regurgitation.* Stenosis or insufficiency of the aortic valve as well as bodily states which are attended by marked hypertension of the greater circulation, such as general arteriosclerosis, chronic interstitial nephritis, and cirrhosis of the liver, are very productive of regurgitation at the mitral valve.

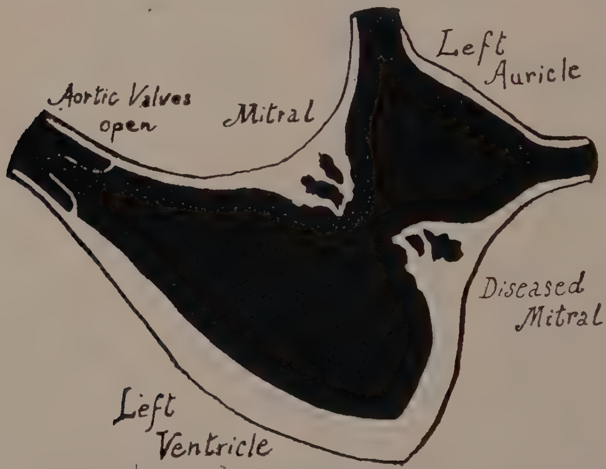

Fig. 313.—Mitral regurgitation. Mitral and aortic valves open during ventricular systole.

The mechanical influences of mitral regurgitation are *primary,* as they are exerted upon the myocardium, and *secondary* as they affect more remote organs and tissues of the body. The clinical manifestations of these influences upon the circulatory imbalance which is induced by the mitral lesion vary in direct proportion to the degree of incompetence and with the extent to which the disturbance in function is compensated by hypertrophy of the myocardium.

During the *first stage* of mitral regurgitation the brunt of the lesion is expended upon the left side of the heart. The imperfect coaptation of the mitral cusps permits a variable quantity of blood to regurgitate into the chamber of the left auricle during ventricular systole. At this period of the cardiac cycle, which corresponds to auricular diastole, the left auricle is receiving the discharge of blood which is returned from the lungs by the pulmonary veins. Thus, the auricle becomes the target for two streams of blood, the one entering its chamber from the pulmonary veins, the other regurgitating from the left ventricle. The fusion of the two streams results in the induction of the so-called "fluid veins," with the generation of an endocardial murmur; and under the influence of these factors the surcharged auricle either compensates by its reserve force or undergoes a transient dilatation, which is followed by compensatory hypertrophy of its wall, whereby it discharges an excess of blood into the left ventricle at each auricular systole. But the mitral insufficiency is still there and not all of this increased auricular output is propelled by the left ventricle into the aorta during its systole. On the contrary, at each ventricular systole a portion of the ventricular blood is returned to the auricle; and to compensate for the extra burden which is thrown upon it, the left ventricle first compensates by using the reserve force commonly used to compensate for increased body activity. The prolonged use of this reserve stimulates compensatory hypertrophy.

In this early stage of the disease the organic change is limited to the left side of the heart. During a period which is variable the hypertrophied left auricle is capable of expelling its contents during systole, and during this time there is no embarrassment of the pulmonary circulation and no extra demand is made upon the right ventricle. Similarly, for a time the hypertrophied left ventricle expels approximately the physiologic quantity of blood into the aorta during its systole, and the normal tension of the general circulation is maintained.

After a period varying with the reserve power of the left heart, the *second stage* of the disease ensues, in which the mechanical influences of the mitral lesion are exerted upon the pulmonary circulation and the right ventricle. The hypertrophied left auricle is now no longer capable of completely expelling its contents during systole, and dilatation beyond the power of compensation ensues. The onward progress of the pulmonary circulation is progressively impeded, and a condition of hypertension is established in this circuit, as evinced by accentuation of the pulmonary second sound. The persistent hypertension induces a varying grade of sclerosis of the pulmonary arterial system, which adds further to the burden thrown upon the right ventricle. At the same time chronic catarrhal inflammation of the bronchial mucous membranes is prone to develop.

The right ventricle undergoes compensatory hypertrophy in response to the increased demand for work, a hypertrophy which during a variable period of time is capable of compensating for the leakage at the mitral valve, and one which progressively increases up to a point which is dependent upon the integrity of the tricuspid valve and the reserve power of the myocardium. As long as the right and left ventricles, acting in unison, are capable of propelling an adequate quantity of blood through the pulmonary and the greater .circulations, there is no apparent imbalance between the two systems. But when this equilibrium is disturbed, the right ventricle dilates beyond the power of compensation, and the *third stage* of the disease is ushered in with failure of the right heart and passive congestion of the general venous system.

With the progressive hypertension in the pulmonary circuit, the right ventricle fails through one of two mechanisms. The tricuspid valve, in the presence of the rising intraventricular pressure may yield and permit a portion of the blood to regurgitate into the right auricle during ventricular systole even in the absence of definite yielding of the ventricular wall, constituting the so-called ''safety-valve leak'' of this orifice. If, on the contrary, the tricuspid valve retains its integrity in the presence of the progressive rise in the intraventricular pressure, the point is reached when the right ventricle is no longer capable of surmounting the excessive pressure in the pulmonary artery. Under these circumstances the ventricle is only partially emptied during ventricular systole. To the residual blood in the right ventricle is added the contents of the right auricle at the succeeding auricular systole; and the walls of the surcharged ventricle yield, with enlargement of the auriculo-ventricular orifice. In this manner is induced the terminal primary effect of the mitral lesion, with the establishment of incompetence of the tricuspid valve, constituting *relative tricuspid insufficiency.*

At this stage of the disease the secondary effects of the mitral lesion develop in rapid succession. The venous return from the trunk and extremities, pouring into the right auricle from the venæ cavæ, encounters a distinct resistance in the regurgitant lesion at the tricuspid valve. The ultimate result is a marked diminution of the volume of blood which reaches the arterial circulation and a dangerous increase of the volume of blood contained in the venous system. As a diminished volume of blood is expelled at each systole of the right ventricle, the left ventricle is only partially filled during diastole, and there is a progressive fall in the general arterial tension. On the other hand, as a consequence of the progressive stasis in the venous system, passive congestion of the lungs and abdominal viscera develops together with effusion into the serous sacs and cellular tissues of the body.

Mitral regurgitation is not infrequently associated with stenosis of the valve, particularly in cases of endocarditic origin. In these cases the lesion which narrows the auriculoventricular orifice likewise prevents accurate coaptation of the cusps during ventricular systole.

Regurgitation at the mitral valve is attended by few symptoms in the first stage when compensation is as yet unbroken. Physical exertion is usually followed by moderate shortness of breath or a fit of cough with the expectoration of a little frothy sputum, which occasionally is tinged with blood. In the second and third stages of the disease, owing to the embarrassment of the pulmonary circulation, symptoms of bronchial inflammation develop with various grades of dyspnea, which finally eventuates in frank orthopnea. Cough is now a constant accompaniment of the disease. Expectoration is profuse, the sputum containing numerous desquamated alveolar epithelial cells which contain blood pigment, the so-called "heart-failure cells."

The subject of advanced mitral regurgitation is subject to disturbances of digestion, and with the progressive venous stasis the development of hemorrhoids is frequently an annoying symptom of the case.

Physical Signs.—Mitral regurgitation in the course of its evolution produces a multiplicity of physical signs, which arise in the main as the result of the primary effects of the lesion upon the myocardium and partially as a manifestation of the secondary effects of the incompetence upon the pulmonary and general circulations. In no disease of the heart can the clinician more readily correlate cause with effect or base a more accurate prognosis upon the physical findings than in mitral regurgitation. With the progress of the disease, there is a progressive augmentation in the physical findings, the paucity of physical signs in the first stage of the disease presenting a striking contrast to the profusion of signs and symptoms which are in evidence during the later periods of the disease.

Inspection.—During the first stage of mitral regurgitation, when the changes in the myocardium are limited to the left side of the heart, inspection is apt to prove negative, save for heaving vigorous impulse and a displacement of the cardiac impulse toward the left and downward, as a result of left ventricular hypertrophy. The degree of apical displacement is, however, quite variable. In the adult subject it is not apt to be as extensive as in children, in whom the apex-beat may be encountered external to the left axillary line. Likewise, in young subjects, owing to the elasticity of the thorax, a moderate degree of bulging of the precordia is occasionally induced by the progressive enlargement of the left heart.

During the second stage of the disease, with the development and maintenance of right ventricular hypertrophy, the cardiac impulse is displaced farther toward the left axilla and is visible over a more extensive area toward the median line and the epigastrium. Systolic pulsation of the epigastrium is almost a constant accompaniment of right ventricular hypertrophy. A similar pulsation not infrequently is visible in the second and third intercostal spaces adjacent to the left sternal border, occurring as a result of left auricular dilatation. At this period of the disease the cardiac impulse is powerful and heaving, in marked contrast to the feeble, slapping, and undulatory impulse following upon complete decompensation of the myocardium.

In the third stage of the disease, with the advent of right ventricular dilatation, the discrete and forcible impulse of the heart progressively gives place to a feeble, undulatory pulsation, which is commonly visible along the left sternal border from the third to the sixth intercostal spaces, and frequently also in the epigastrium along the left costal arch. At this time also the positive venous pulse is demonstrable in the jugular veins as a result of tricuspid leakage, and occasionally there is a systolic hepatic pulsation arising as a result of the same lesion.

At the same time signs of general venous stasis appear, as evinced by edema of the feet and ankles, progressive abdominal enlargement from ascites, chronic cough with the raising of serous, sometimes blood-streaked expectoration, together with acceleration of the respiratory movements of the thorax and cyanosis of the mucous membranes, auricles, and digits. In chronic cases of extensive duration clubbing of the fingers is frequently seen, and these subjects often exhibit a tortuosity and distention of the superficial veins of the neck and chest.

Palpation.—During the maintenance of left ventricular hypertrophy in the first stage of the disease the cardiac impulse is discrete, forceful, and heaving upon palpation of the precordia. In a relatively small percentage of cases of pure mitral regurgitation a fine systolic thrill is to be detected over the cardiac apex. A thrill is, however, much more commonly encountered in the cases of combined stenosis and regurgitation at the mitral orifice.

In the second stage of the disease, with the development of compensatory hypertrophy of the right ventricle, the palpable impulse of the heart becomes more extensive, while preserving its force. Systolic lifting of the lower costal margin and upper epigastrium is frequently to be detected, as well as a systolic shock communicated to the liver by the overacting right ventricle.

With the progressive failure of the right ventricle during the third stage of the disease the cardiac impulse becomes progressively enfeebled, diffuse, and wavy, frequently extending to the right of the sternum and well into the epigastrium. Positive systolic pulsation of the jugular veins is to be detected by compression of the vein just above the clavicle, a simple maneuver which abolishes a true systolic jugular pulsation and thus serves to distinguish it from a false pulsation communicated to the vein from the subjacent carotid artery. Bimanual palpation frequently reveals a systolic liver pulsation.

The *pulse* in mitral regurgitation is to a large degree indicative of the state of the left ventricle throughout the course of the disease. So long as compensation is maintained, the radial pulse is of constant volume and tension and its rhythm is usually not disturbed. Moderate acceleration is frequently present, however, and in the presence of auricular fibrillation, arrhythmia is a striking feature throughout the course of the disease. With the supervention of left ventricular dilatation the volume of the pulse is markedly reduced, acceleration is more marked, and irregularity of both force and rhythm is usually demonstrable. In the third stage of the disease not every ventricular systole may be sufficiently powerful to produce a radial pulse.

Percussion.—The transverse dullness of the heart is increased toward the left and downward as a consequence of the bilateral ventricular hypertrophy which attends the disease. So long as hypertrophy is limited to the left side of the heart, the outline of cardiac dullness at the apex remains pointed; but in the second stage of the disease, with the establishment of right ventricular hypertrophy, there is a progressive rounding of the cardiac outline at the apical area, while the dullness extends farther to the right of the sternum. In the late period of the disease, with the establishment of tricuspid insufficiency, the examiner may frequently detect an extension of cardiac dullness upward in the third and fourth intercostal spaces upon either side of the sternum, as a result of auricular dilatation.

The pulmonary bases should be carefully and methodically percussed during every examination of a subject of mitral regurgitation with the object of detecting a complicating pulmonary edema or hydrothorax. This examination commonly proves negative during the period of maintained compensation during the first and second stages of mitral regurgitation. But with the rupture of compensation in the third stage of mitral insufficiency percussion of the pulmonary bases yields impairment of vesicular resonance due to pulmonary edema, or flatness over the inferior portion of the thorax as a result of hydrothorax.

Similarly, a routine examination in this disease should not be considered complete without the careful delimitation of the areas of hepatic and splenic dullness. With the maintenance of compensation in the first and second stages of the disease these areas of dullness retain their normal dimensions; but with failure of compensation and the general venous stasis which attends this state the areas of hepatic and splenic dullness are increased in extent as a result of passive congestion of the venous radicles of these organs.

Auscultation.—Mitral regurgitation in the first period of its evolution is attended by a systolic murmur with its point of maximum intensity at the mitral area, over the apex of the heart. From this site the murmur is propagated in all directions; but it is transmitted with selective intensity toward the left axillary region; and occasionally it is audible upon the posterior aspect of the thorax near the angle of the scapula. The murmur is blowing, sometimes harsh and usually unmusical, and it partially or completely obscures the first sound of the heart at the apex. The quality and intensity of the murmur vary with the condition of the myocardium and with the posture assumed by the patient. A murmur which is faintly audible in the erect posture frequently becomes accentuated when the subject assumes the recumbent posture. During the first stage of mitral regurgitation there is no perceptible accentuation of the second sound of the heart at the pulmonic area, a negative finding which, by excluding the existence of right ventricular hypertrophy, indicates that the myocardial changes are as yet limited to the left heart.

During the second stage of the disease, on the contrary, accentuation of the pulmonic second sound is clearly perceptible, affording a perfectly reliable sign of compensation of the mitral lesion by right ventricular hypertrophy. But before basing deductions as to the extent of the mitral lesion upon the degree of accentuation of the pulmonic sound, the examiner must needs exclude the presence of obstructive disease of the lungs, and must take into consideration the age of the patient, bearing in mind the physiologic accentuation of this sound in the young subject.

During the first and second stages of the disease auscultation of the pulmonary bases yields no signs of congestion of the lungs or pulmonary edema; and so long as the mitral leakage is adequately compensated by right ventricular hypertrophy, the intensity of the mitral murmur remains uniform in intensity, in striking contrast to the enfeeblement of the murmur which is encountered in the last stage of the disease.

With the failure of compensation by the right ventricle there is a progressive enfeeblement of the accentuated pulmonary second sound, with the coincident generation of the systolic murmur of a ''safety-valve leak'' at the tricuspid orifice. As the systolic murmur of tricuspid insufficiency with its point of maximum intensity over the lower sternal region gains in intensity, there is a progressive enfeeblement of the mitral systolic murmur, which is audible over an ever-diminishing area, to finally become masked entirely by the increasing intensity of the tricuspid murmur.

In this late stage of the disease auscultation of the pulmonary bases yields numerous moist râles, which are universally distributed over both lungs.

Diagnosis.—The presence of a systolic murmur at the cardiac apex usually blowing and unmusical in quality, which is transmitted toward the left axilla, is suggestive of the murmur of mitral regurgitation. Before such a diagnosis is made, however, the examiner should seek such signs of ventricular hypertrophy as displacement of the impulse downward and toward the left and accentuation of the pulmonic second sound, as the mere presence of a systolic bruit at the mitral area is not sufficient evidence for a diagnosis of mitral regurgitation. The elicitation of a history of rheumatic fever, tonsillitis or chorea, combined with a poor response to exercise tests, speaks in favor of organic disease of the valve.

Differential Diagnosis.—*Functional murmurs* are prone to develop at the mitral valve, although they are more frequently encountered at the pulmonic valve. Nevertheless, the examiner should bear in mind that the mitral valve comes second in point of frequency for these murmurs. But, as previously noted, these murmurs do not produce ventricular hypertrophy neither are they propagated beyond the limits of the precordia, and usually they are encountered in the anemic and debilitated subject.

The systolic murmur of *aortic stenosis* is frequently audible at the mitral area as well as at the aortic area; but in this instance the examiner will experience little difficulty in determining that the point of maximum intensity of the murmur is localized in the aortic area, and that its line of propagation is upward into the root of the neck and not at all toward the left axillary region. Moreover, the quality of the two murmurs is quite dissimilar. The murmur of aortic stenosis is loud and harsh as compared with the softer ''whiff'' of the mitral regurgitant murmur, and aortic stenosis is almost constantly attended by a palpable thrill in the aortic valve area.

Pericardial friction is frequently audible over the region of the cardiac apex, producing in this locality a sound with a quality not unlike that of the mitral regurgitant murmur. But while pericardial friction is frequently almost synchronous with the first sound of the heart, the sound is more diffuse in its distribution, and it is never transmitted from the limits of the precordia. Neither does the sound completely replace the first sound of the heart; and moreover, it is frequently transient and evanescent and is influenced by pressure with the stethoscope and by changes in posture upon the part of the patient.

A *cardiorespiratory murmur* generated near the cardiac apex may readily be mistaken for a mitral systolic murmur during a casual examination; but this murmur does not replace the first sound of the heart; and it is markedly influenced by the respiratory excursion of the lung, being intensified during full inspiration, and frequently abolished during forced expiration.

MITRAL STENOSIS

Clinical Pathology.—Stenosis of the mitral valve is usually a disease of adolescence and early adult life, developing as a sequence of acute endocarditis of rheumatic origin. Aside from the rheumatic cases may be grouped endocarditic cases arising as a sequence of scarlatina, chorea, chlorosis, and the acute infectious fevers. But there is another group of cases of mitral stenosis in which a slow sclerosis of the valvular cusps occurs in elderly subjects of general arteriosclerosis and chronic interstitial nephritis is said to be more common in women due to the fact that chorea is more common in girls.

The mechanical influences of the lesion in mitral stenosis are manifested in much the same manner as are those which accompany a regurgitant lesion at this valve, with certain variations which are due to the manner of inception of the primary alterations in the myocardium. As these influences operate slowly upon the myocardium and remote organs of the body, the disease may be divided into three stages, as in the case of mitral regurgitation, three stages in which one results inevitably in another when once an organic stenosis is established at the mitral orifice.

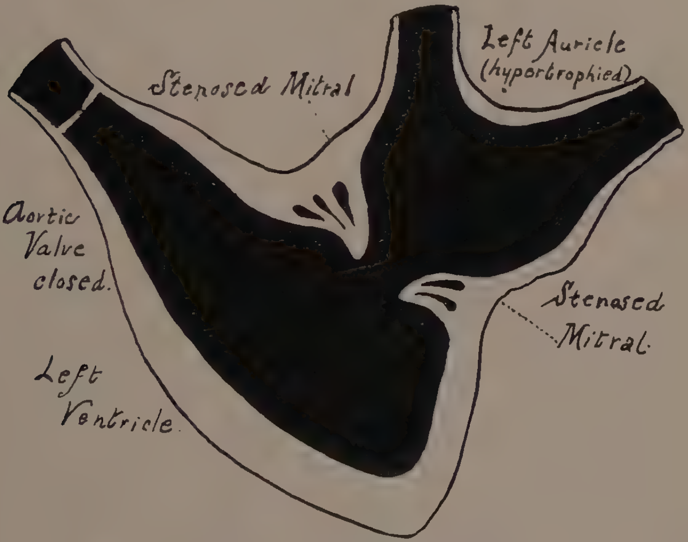

Fig. 314.—Mitral stenosis. Ventricle during late diastole or presystole.

During the first stage the brunt of the lesion falls upon the left auricle, and during this stage of the disease the myocardial changes are confined to the left side of the heart. At the completion of ventricular systole the ventricular walls relax and during the early portion of ventricular diastole a portion of the blood which has accumulated in the left auricle during ventricular systole is aspirated into the chamber of the left ventricle. During the midportion of ventricular diastole the pressure of the blood column contained in the left auricle and the pulmonary veins forces a further quantity of the contents of the left auricle into the chamber of the left ventricle. Finally, during late ventricular diastole the systole of the left auricle propels the remainder of the blood which occupies the cavity of the auricle into the ventricle, completing the first act of the cardiac cycle. In the presence of a narrowing of the mitral orifice an additional burden is thrown upon the left auricle, which hypertrophies in response to the increased demand for work. So long as the hypertrophied auricle is capable of completely emptying its contents promptly without retardation of the pulmonary circulation, the circulatory balance is adequately maintained; but as the reserve power of the auricle is limited, hypertrophy eventually gives way to dilatation, an increased burden is thrown upon the pulmonary circulation and the right heart, and the second stage of the disease is ushered in with hypertrophy of the right ventricle. During this stage of the disease moderate left ventricular hypertrophy occurs as the result of the aspirating action of this chamber in drawing its proper quota of blood through the narrowed orifice, and because it must contract powerfully in order to expel the diminished output of the auricle into the aorta.

The third stage of the disease develops when the right ventricle yields to the progressive increase in tension of the pulmonary circuit, the mechanism differing in no wise from that of mitral regurgitation.

The valvular changes in mitral stenosis present wide variations. In early endocarditic cases there are merely small vegetations upon the auricular aspects of the cusps, which produce practically no stenosis, but which are sufficient to induce "fluid veins" with the generation of a presystolic murmur. In more advanced cases the cusps are sclerosed and shrunken, frequently with adhesion of the free borders and shortening of the papillary muscles, with the reduction of the valvular orifice into a mere slit or chink. In yet other instances the cusps are normal, but the mitral ring is reduced in caliber either as a congenital defect or as a result of secondary sclerosis.

During the first and second stages of mitral stenosis the subject of the disease experiences little discomfort and presents few symptoms. Foremost among these is moderate dyspnea which is brought out upon severe exertion. Occasionally the first intimation of the existence of the disease consists in the onset of a local paralysis from embolism of the brain. In recurrent endocarditic cases the subject is likely to present recurrent febrile attacks which develop without assignable cause. During the later stage of the disease dyspnea becomes extreme, amounting frequently to orthopnea, with free expectoration of frothy sputum which is frequently tinged with blood.

Physical Signs.—*Inspection.*—In the first stage of mitral stenosis inspection frequently proves negative, as the principal findings upon inspection in this disease depend upon embarrassment of the pulmonary circulation as a result of the mitral obstruction, and as this obstruction is adequately compensated during the early period of the disease by left auricular hypertrophy. The cardiac impulse usually occupies its normal position in the fifth left interspace; and when it is displaced by left ventricular hypertrophy at this time the displacement occurs toward the left and downward. The area of the impulse is very frequently reduced in extent. In the case of young subjects and in women there is frequently a visible presystolic pulsation in the second and third intercostal spaces adjacent to the left sternal border, which is produced by left auricular hypertrophy.

During the second stage of the disease, with the establishment of right ventricular hypertrophy, systolic pulsation of the epigastrium is frequently to be noted, as well as a wide systolic pulsation in the third and fourth intercostal spaces along the left sternal border, developing as a result of the impact against the thoracic wall of the conus arteriosus of the right ventricle. In children and in thin chested subjects a variable degree of bulging of the precordia and lower sternal region is rarely encountered.

With failure of the right ventricle in the third stage of the disease the cardiac impulse becomes broad and undulatory along the left sternal border, together with distention of the cervical veins and frequently a positive jugular pulsation which is due to the establishment of tricuspid regurgitation. The respiratory movements of the thorax are labored, and the lips and mucous membranes exhibit cyanosis together with general pallor of the integument of the trunk and extremities. The abdomen protrudes from the accumulation of fluid in the peritoneal cavity, and the feet and ankles swell from edema of the cellular tissues.

Palpation.—Palpation of the mitral area in mitral stenosis reveals the presence of a presystolic thrill, which is pathognomic of the disease. The apex beat is of normal or slightly increased force. The thrill at the mitral area is attended by accentuation of the valve shock at the pulmonic area, which attains its maximum intensity during hypertrophy of the right ventricle during the second stage of the disease. During the third stage of the disease the mitral thrill becomes progressively enfeebled while the cardiac impulse becomes more extensive, undulatory and slapping.

The pulse of mitral stenosis is of small volume, but of high tension during the maintenance of compensation, and is disordered in both force and rhythm when auricular fibrillation develops. In fibrillation cases arrhythmia is a striking feature throughout the course of the disease.

Percussion.—During the first stage of mitral stenosis percussion reveals no alteration in the cardiac outline, save in selected cases an extension of dullness in the third and fourth left interspaces arising from left auricular hypertrophy, and to the left and downward if left ventricular hypertrophy occurs. During the second stage of the disease the area of cardiac dullness is extended toward the right and left, encroaching for a variable distance upon the vesicular resonance of Ebstein's cardiohepatic angle. During the third stage of the disease percussion of the pulmonary bases frequently reveals dullness from pulmonary edema; and the areas of hepatic and splenic dullness are extended as a result of chronic passive congestion of these organs.

Auscultation.—Mitral stenosis is attended by a presystolic or a diastolic murmur, according to the duration of the disease. The initial murmur to appear is a presystolic bruit, which has its point of maximum intensity a little to the right of the cardiac apex, and which is not transmitted to any appreciable distance from this site.

The presystolic murmur of early mitral stenosis possesses a harsh quality, and it is ingravescent or crescendo in character, increasing steadily in intensity from commencement to termination, and is followed by a sharp and snapping first sound of the heart in the absence of coincident mitral regurgitation. In cases of minor degrees of stenosis of the valve, however, in which a few small vegetations exist upon the auricular aspect of the valve segments, there may be no distinct murmur, but merely a roughening and accentuation of the first sound of the heart at the apex. When the disease is complicated by auricular fibrillation, the presystolic murmur disappears with the onset of this abnormal rhythm.

During the later stages of the disease a diastolic murmur is commonly audible, which has its inception immediately after the completion of the second sound. At the time of its appearance this murmur assumes a diminuendo character, occupying only the early portion of diastole and soon dying away. In certain instances, however, the diastolic murmur is merged into a concomitant presystolic murmur, with the consequent production of a continuous murmur, which persists throughout the diastolic period. This composite murmur is quite intense at its inception; it then diminishes in intensity; and it finally terminates in an ingravescent or crescendo presystolic phase. Upon failure of the left auricular musculature, the presystolic element of the murmur is lost, but the diastolic portion remains audible, consuming a portion or the whole of the diastolic phase.

During the period of right ventricular hypertrophy the second sound at the pulmonary area exhibits moderate accentuation. With the persistent hypertension in the pulmonary circulation, however, there is a gradual yielding of the pulmonary valve, with the later induction of the Graham Steell murmur with its point of maximum intensity in the third left interspace adjacent to the sternum.

As mitral stenosis and regurgitation are frequently concomitant diseases, the murmur of mitral stenosis is frequently followed by the systolic murmur which is produced by the regurgitant lesion.

In young subjects the rhythm is apt to prove normal over a prolonged period. In late cases, however, auricular fibrillation frequently occasions a totally irregular rhythm, which tends to persist.

Electrocardiogram.—The electrocardiogram often shows a large *P* wave. This is also frequently notched. Such *P* waves are by no means confined to instances of mitral stenosis. The electrical axis frequently deviates to the right. It often shows no such deviation, and may even incline to the left. The electrocardiogram is of some confirmatory value, but its importance has been overestimated.

Diagnosis.—The diagnosis of mitral stenosis is based upon the characteristic physical findings; namely, a presystolic murmur at the mitral area, ingravescent or crescendo in quality, followed by a distinct and snapping first sound of the heart, and not transmitted beyond the precordia, with increase in the transverse dullness of the heart, particularly toward the right, and with accentuation of the pulmonary second sound at the base of the heart. There are, however, a number of possible confusing findings, and a differential diagnosis is often attended by considerable difficulty.

Differential Diagnosis.—Foremost among the confusing factors in the diagnosis of mitral stenosis stands the *Flint murmur* of aortic regurgitation. In the exclusion of this possible source of error the examiner should search for the aortic organic murmur occurring at the base of the heart in this disease, the capillary pulse of Quincke and the femoral murmur of Duroziez. The quality of the two murmurs is quite dissimilar, as the Flint murmur does not possess the ingravescent and crescendo quality of the murmur of true organic mitral stenosis. While both murmurs occur at the mitral area and are not transmitted thence, the Flint murmur never possesses the same degree of intensity as is attained by the murmur of mitral stenosis.

Mitral regurgitation is attended by a soft, blowing murmur at the mitral area, which is systolic in time, and which is transmitted toward the left axilla. The progress of development of the two murmurs is, moreover, diametrically opposed, the murmur of mitral stenosis progressively increasing in intensity to terminate in an abrupt first sound of the heart, whereas the murmur of mitral regurgitation, becomes progressively less intense from its commencement with ventricular systole until its termination. Quite frequently the two murmurs are combined, the systolic murmur of mitral regurgitation ensuing shortly upon the presystolic murmur of mitral stenosis, in the presence of combined stenosis and regurgitation at the mitral orifice.

Tricuspid regurgitation produces a systolic murmur with its point of maximum intensity in the tricuspid area over the lower portion of the sternum, whence the murmur is propagated toward the right. This murmur frequently is audible in combination with the murmur of mitral stenosis late in the course of the disease; but it is attended by such striking disorders of the venous circulation that a differentiation of the two murmurs is not attended by great difficulty.

Tricuspid stenosis produces a presystolic murmur with its point of maximum intensity not far removed from that of the murmur of mitral stenosis. The quality of the two murmurs is not dissimilar, but stenosis of the tricuspid orifice is a rare lesion and usually forms a part of the syndrome of congenital cardiac disease.

The murmur of *aortic regurgitation* occasionally has its point of maximum intensity at the mitral area; but in this instance the time of the murmur as well as the quality serves as a basis of differentiation. The murmur of aortic regurgitation has its inception at the commencement of ventricular diastole, progressively becoming less intense during the diastolic period to become lost before the beginning of the first sound of the heart. In those cases of mitral stenosis, however, in which the mitral presystolic murmur occupies a considerable portion of the diastolic period, an error in diagnosis is very prone to be made.

PULMONARY REGURGITATION (PULMONARY INSUFFICIENCY; PULMONARY INCOMPETENCE)

Clinical Pathology.—Pulmonary regurgitation occurring as the result of organic disease of the pulmonary valve is the rarest of all acquired lesions of the heart. Isolated cases are encountered, however, in which as the result of ulcerative endocarditis attacking the right side of the heart, deformity of the valvular cusps has occurred with resulting inability of the deformed cusps to adequately close the pulmonary orifice. These cases are most frequently encountered among young adult subjects, and pulmonary insufficiency is usually combined with pulmonary stenosis. Incompetence of the pulmonary valve is one of the rare congenital defects of the heart.

Relative pulmonary regurgitation, on the contrary, in which regurgitation occurs at this orifice in the absence of structural derangement of the valve, is of frequent occurrence. Relative pulmonary insufficiency may be of intracardiac or extracardiac origin. In the presence of uncompensated lesions of the left side of the heart, with the progressive rise in tension in the pulmonary circulation, the right ventricle is prone to yield with resulting enlargement of the ring supporting the cusps of the semilunar valve and the induction of relative pulmonary insufficiency. Similarly, in the presence of obstructive disease of the lungs of prolonged duration, such as chronic ulcerative phthisis, cirrhosis of the lung, and hypertrophic emphysema, the persistent hypertension in the pulmonary circulation is capable of inducing relative insufficiency of the pulmonary valve.

The immediate effect of regurgitation at the pulmonary valve is to induce transient dilatation of the right ventricle, which is compensated by hypertrophy of its muscular walls. So long as compensation is maintained the circulatory imbalance is corrected; but the ventricle eventually reaches the limit of its reserve power, whereupon dilatation beyond the power of compensation follows, with the establishment of tricuspid regurgitation, and general engorgement of the venous system. As a result of stasis in the pulmonary circulation edema of the lungs ensues and the subject exhibits the signs of chronic bronchial catarrh.

Physical Signs.—*Inspection.*—In its early stages simple pulmonary regurgitation yields no evidence of its existence upon inspection. As the disease progresses the cardiac impulse is displaced toward the left axillary line but is not displaced downward. While right ventricular hypertrophy is maintained, there is visible pulsation of the epigastrium, and in thin chested individuals there may be visible bulging of the lower portion of the precordia. As the disease progresses the subject becomes dyspneic with chronic cough and serous expectoration, while the trunk and extremities are pallid or cyanotic as a result of deficient arterial circulation. With the establishment of tricuspid insufficiency the positive jugular pulse becomes demonstrable in the jugular veins.

Palpation.—During the early period of the disease with the maintenance of right ventricular hypertrophy the apex beat is diffuse and extends downward toward the epigastrium and well inward toward the left sternal border. A forceful, lifting systolic pulsation is usually to be elicited in the upper epigastrium as a result of the powerful contractions of the right ventricle. Similarly, the right costal arch is occasionally raised by the impact of the overacting right ventricle against the liver. Late in the disease a true expansile pulsation of the liver can occasionally be elicited upon bimanual palpation of the liver, occurring as a manifestation of tricuspid regurgitation. Occasionally it is possible to elicit a fine diastolic thrill at the pulmonary area; but the thrill is not constantly present, neither is it detected with the same constancy as in the case of the systolic thrill accompanying stenosis of this valve.

The *pulse* in pulmonary regurgitation is apt to be of low tension and diminished volume and arrhythmic, but presents nothing which is characteristic of the disease.

Percussion.—The transverse dullness of the heart is increased toward the right in direct proportion to the degree of hypertrophy of the right ventricle. As dilatation ensues upon compensatory hypertrophy, the right margin extends well to the right of the sternum. Late in the disease the pulmonary resonance is impaired over the bases of the lungs, and the areas of dullness of the liver and spleen are more extensive than in the normal subject.

Auscultation.—Auscultation of the pulmonary area reveals the presence of a blowing murmur with its point of maximum intensity at the junction of the second left costal cartilage with the sternum, whence it is transmitted downward along the left sternal border and toward the cardiac apex. The murmur is commonly blowing and musical, but in cases of associated pulmonary stenosis and regurgitation the murmur is apt to have a harsher quality and to completely replace the second sound of the heart at the pulmonary area. In the presence of relative pulmonary insufficiency auscultation of other valve areas will elicit the murmurs peculiar to the organic lesions which are present.

Diagnosis.—The diagnosis rests upon the detection of a diastolic murmur with its point of maximum intensity at the pulmonary area, whence it is transmitted downward, with signs of hypertrophy or dilatation of the right heart, and general venous stasis. In reaching a diagnosis of simple pulmonary regurgitation the examiner should bear in mind the relative infrequency with which this valve is attacked by organic disease.

PULMONARY STENOSIS

Clinical Pathology.—Stenosis at the pulmonary valve is in the vast majority of cases due to a congenital defect and is associated with other congenital deformities of the heart such as tricuspid stenosis, patent ductus arteriosus, patent foramen ovale, or perforation of the ventricular septum. Very rarely the lesion is the sequence of ulcerative endocarditis attacking the pulmonary valve.

But while true stenosis of the pulmonary valve is a rare lesion, there are many pathologic conditions capable of inducing the generation of a systolic murmur at this valve. First and foremost among these factors stands anemia with the induction of functional murmurs, which are more frequently encountered at the pulmonary valve than at the other valves of the heart. Similarly, dilatation of the pulmonary artery distal to the valve produces a condition of *relative pulmonary stenosis,* which is attended by a systolic murmur at this area in the absence of organic disease of the valve. Compression or constriction of the pulmonary artery by new growths, enlarged glands, or pleural adhesions are all capable of inducing a murmur which may be mistaken for that of pulmonary stenosis. The nervous imbalance of the cardiac mechanism incident to exophthalmic goiter frequently is betrayed by a systolic murmur at the pulmonary valve; and shifting of the mediastinal structures through cardiac displacement may produce torsion of the vessel with a resulting systolic murmur in the vicinity of the pulmonary orifice.

The immediate effects of the lesion in stenosis at the pulmonary valve are expended upon the right ventricle, which hypertrophies to compensate for the obstruction; and if the obstruction is slight, no further myocardial change may be induced. In the presence of a greater degree of stenosis however, the persistent increase of intraventricular pressure results in right ventricular dilatation, relative tricuspid insufficiency and general venous engorgement.

Physical Signs.—*Inspection.*—In cases of pulmonary stenosis of congenital origin, the signs upon inspection are largely those of congenital heart disease, notably extreme cyanosis, dyspnea and and clubbing of the fingers. In the presence of minor degrees of stenosis, dyspnea and a little cyanosis upon active exertion may remain the sole signs of the disease. When right ventricular hypertrophy is a marked feature of the case the cardiac impulse is displaced toward the left and is visible also farther toward the right as a systolic pulsation of the upper portion of the epigastrium. With the supervention of tricuspid insufficiency the positive venous pulse becomes appreciable in the jugular veins.

Palpation.—Palpation of the pulmonary valve area reveals the presence of a fine systolic thrill, which is frequently diffused over a wider area than is the case with the systolic thrill of aortic stenosis. The thrust of the right ventricle is clearly palpable in the upper epigastrium in the presence of hypertrophy of this chamber of the heart, and its impact not infrequently communicates a systolic impulse to the adjacent liver.

The *pulse* at no time during the course of the disease presents any changes which are characteristic of the disease. With failure of the right heart its volume is reduced; this is in no wise distinctive of the lesion present.

Percussion.—Delimitation of the cardiac outline proves negative during the early stages of pulmonary stenosis, and in the presence of minor degrees of stenosis which are readily compensated by a slight degree of right ventricular hypertrophy the outline is little altered throughout the disease. With the supervention of frank ventricular hypertrophy, the outline is increased toward the right; and in the presence of right ventricular and auricular dilatation the increase toward the right is further accentuated.

With the establishment of tricuspid regurgitation and venous stasis the dullness of the liver and of the spleen is appreciably increased in extent.

Auscultation.—Pulmonary stenosis is attended by a systolic murmur with its point of maximum intensity at the pulmonary area or an interspace below, whence it is diffused in all directions, but with selective intensity upward toward the root of the neck, but it does not extend into the vessels of the cervical region as is the case with the systolic murmur of aortic stenosis. The murmur is audible over a considerable area of the precordia, but with care its point of maximum intensity can be localized to the second or third left intercostal space adjacent to the sternum.

The quality and intensity of the murmur vary with the degree of stenosis and with the power of the ventricular contraction. The murmur is inherently harsh and unmusical, in marked contrast to the soft and blowing murmurs of functional origin occurring at this valve. In the presence of minor grades of stenosis, however, the intensity of the murmur is not great and its duration is not considerable. In the case of marked stenosis, on the contrary, the murmur is harsh, intense, and sustained, commencing with ventricular systole and terminating with the second sound of the heart, which is not infrequently replaced by the diastolic murmur of a coexistent pulmonary regurgitation.

With the establishment of tricuspid insufficiency, the systolic murmur of this lesion is perceptible at the tricuspid area, obscuring in this locality the first sound of the heart.

Diagnosis.—The diagnosis of simple pulmonary stenosis rests upon the cardinal signs of the disease; namely, a systolic murmur with its point of maximum intensity at the pulmonary area, whence it is transmitted upward, but which does not extend into the great vessels of the neck; a palpable thrill at the same area, which is to be detected in the vast majority of cases; and signs of right ventricular hypertrophy or of right heart failure.

Differential Diagnosis.—*Functional murmurs,* which are encountered at the pulmonary valve more frequently than at the other valve areas of the heart, are systolic in time; but they are soft and blowing; they are transient, coming and going at successive examinations; they are not transmitted beyond the limits of the precordia; and they do not induce changes in the myocardium or the character of the pulse.

In the *spurious murmurs* which are generated at this area by torsion or dilatation of the pulmonary artery, by the constriction of the vessel by pleural adhesions, or by the disturbance of exophthalmic goiter, there is no evidence of circulatory disturbance or of myocardial changes. The patient is not dyspneic and is apparently in a good state of health. The murmurs, moreover, are more diffuse than is the murmur of simple pulmonary stenosis, and they exhibit no selective line of transmission.

Aneurysm of the aorta produces a systolic murmur which is audible in the vicinity of the pulmonary area; but the murmur and the thrill which accompanies it has a more diffuse distribution upon the anterior thoracic surface; it is commonly attended by local pulsation and by dullness over its distribution, and its systolic murmur is transmitted into the vessels of the neck, which is not true of the murmur of pulmonary stenosis.

The x-ray will usually demonstrate the tumor in aneurysm.

Patency of the ductus arteriosus induces a murmur in the vicinity of the pulmonary area; but the point of maximum intensity of this murmur is farther from the sternal margin than is the case with the pulmonary stenotic murmur; and, moreover, the murmur of a patent ductus arteriosus continues beyond the second sound of the heart, which is not characteristic of the murmur of pulmonic stenosis.

TRICUSPID REGURGITATION (TRICUSPID INSUFFICIENCY; TRICUSPID INCOMPETENCE)

Clinical Pathology.—Regurgitation occurs at the tricuspid orifice in two distinct forms; namely, as simple organic insufficiency of the valve, and as relative or functional insufficiency of the tricuspid valve. Organic disease of this valve of the heart is indeed rare, as the predilection of endocarditic changes is for the left side of the heart. Occasionally tricuspid regurgitation arises from congenital malformation of the valve, in which event it is usually combined with tricuspid stenosis.

If organic insufficiency at the tricuspid valve is infrequent, relative regurgitation at this orifice, arising as a result of right ventricular dilatation under the stress of heightened tension in the pulmonary circulation, is a very frequent disease. Under these circumstances the tricuspid valve develops a "safety-valve leak" with the generation of a soft and blowing systolic murmur with its point of maximum intensity at the tricuspid area over the lower end of the gladiolus, the valvular disease constituting merely a link in the syndrome of chronic valvular disease of the heart. Indeed, even in the absence of incompetence upon the part of the valves of the left heart, tricuspid regurgitation may be induced by persistent hypertension in the pulmonary circulation, which arises as a result of obstructive disease of the lungs during the course of chronic ulcerative phthisis, chronic interstitial pneumonia, or hypertrophic emphysema.

The primary effect of the tricuspid lesion is an immense dilatation of the right auricle, the reserve power of which is so limited that auricular hypertrophy never adequately compensates for the lesion at the tricuspid valve. Venous engorgement appears early in the disease, with overfullness of the cervical veins, systolic pulsation of the liver, the positive venous pulse in the jugulars, anasarca and general edema of the extremities.

Physical Signs.—*Inspection.*—In tricuspid regurgitation the physical signs vary with the state of the myocardium and with the syndrome of which tricuspid regurgitation is a part. In the presence of simple isolated tricuspid regurgitation of congenital or endocarditic origin the cardiac impulse is carried toward the right as a result of right ventricular hypertrophy, sometimes occupying a position behind the sternum. In relative tricuspid insufficiency occurring as part and parcel of left-sided valvular disease, on the contrary, the cardiac impulse is displaced to the left and downward, at the same time encroaching upon the upper epigastrium, in which site it is heaving and forceful if right ventricular hypertrophy is maintained. As the disease progresses, dilatation of the right auricle produces a visible pulsation along the right sternal border in the second, third, and fourth interspaces.

Whatever may be the cause of the lesion, tricuspid insufficiency produces multiple signs of venous engorgement and general venous stasis. The subject is dyspneic and frequently cyanotic about the face, with anemic pallor of the trunk from deficient arterial circulation. The positive venous pulse is demonstrable in the jugular veins, and the abdomen is prominent from ascites, while the feet and ankles swell from edema.

Palpation.—Palpation of the upper epigastrium reveals a systolic pulsation, arising as the result of the impact of the right ventricle. In simple organic regurgitation of the tricuspid valve this pulsation does not possess the same degree of force which is exhibited in the secondary or relative form of the disease, in which right ventricular hypertrophy is more extreme. Bimanual palpation of the liver reveals the presence of a true, expansile pulsation of this organ which is synchronous with ventricular systole. A pseudopulsation of the liver is sometimes caused by the impact of the hypertrophied right ventricle against the liver, and should not be mistaken for the true expansile pulsation of the organ.

The *pulse* of tricuspid regurgitation presents no distinctive characteristics. Asymmetry of the radial pulses is apt to arise as the result of compression of the right subclavian artery by the enlarged right auricle.

Percussion.—In simple tricuspid regurgitation, the cardiac outline extends well to the right of the sternum, with little or no enlargement of the left ventricle. In relative tricuspid regurgitation, on the contrary the transverse dullness of the heart is extended both toward the right and toward the left and downward toward the upper epigastrium as a result of combined ventricular hypertrophy or dilatation.

Percussion of the base of the thorax reveals impairment of the vesicular resonance as a result of pulmonary edema or frank dullness late in the disease from hydrothorax. The areas of hepatic and splenic dullness are extended in the late stages of the disease.

Auscultation.—Tricuspid regurgitation is attended by a blowing systolic murmur with its point of maximum intensity at the tricuspid area over the lower portion of the gladiolus, whence it is transmitted toward the right and slightly upward or to the right and downward into the liver. The point of maximum intensity of this murmur is not as discrete and circumscribed as is the case with endocardial murmurs occurring at the other valves of the heart. Frequently the blowing murmur is audible over the greater portion of the precordia; but careful examination will usually serve to localize its maximum intensity over the lower sternal region and to reveal a selective transmission of the murmur toward the right and upward. The first sound of the heart is obscured by the murmur, but is seldom entirely replaced by it. The second sound varies with the concomitant state of the myocardium. In simple isolated tricuspid regurgitation the second sound is unaltered in intensity or slightly enfeebled, whereas in relative tricuspid insufficiency the second sound is accentuated during the maintenance of right ventricular hypertrophy, to become enfeebled with the supervention of right ventricular dilatation.

Diagnosis.—The cardinal signs of tricuspid regurgitation are a systolic murmur with its point of maximum intensity at the tricuspid area, whence it is transmitted toward the right and slightly upward or downward into the liver, signs of right ventricular hypertrophy, the positive venous pulse in the jugulars, the systolic hepatic pulse, and signs of general venous engorgement. In cases of relative tricuspid regurgitation the murmurs of the causative left-sided valvular lesions are to be detected.

TRICUSPID STENOSIS

Clinical Pathology.—Stenosis at the tricuspid orifice ranks among the rarest of the valvular lesions of the heart. The majority of the cases have occurred in female subjects, and the lesion is rarely diagnosed during life.

Most cases of tricuspid stenosis are of congenital origin, the segments by fusion of their free borders leaving a mere buttonhole or chink at the tricuspid orifice. There are cases, however, which are of endocarditic origin, and which are associated with similar stenotic lesions at the mitral orifice.

The primary effect of the stenotic lesion is to add to the burden of the right auricle, which hypertrophies in response to the increased demand for work. In cases of moderate stenosis of the valve this compensatory hypertrophy is adequate and is maintained and there is no embarrassment of the venous circulation. In more pronounced cases, however, right auricular dilatation eventually supervenes, with signs of general venous stasis, and effusion into the serous sacs and the cellular tissues of the body.

Physical Signs.—*Inspection.*—Many cases of tricuspid stenosis with adequate compensation yield no physical signs for years or those which are exhibited are obscure and conflicting. Signs which point to stenosis of the tricuspid orifice comprise persistent moderate cyanosis, with distention and tortuosity of the cervical veins, and occasionally a pronounced negative venous pulse in the jugular veins.

Palpation.—Tricuspid stenosis is occasionally attended by a presystolic thrill which is palpable at the tricuspid area over the lower end of the sternum, but the thrill is not a constant finding in this disease. The cardiac impulse is usually encountered in its normal site, and its force and extent are not deranged in many cases. Pronounced stenosis of the valve, however, causes diminution in the force and extent of the apex-beat, and its rhythm is apt to be deranged.

The *pulse* is in no wise characteristic of the disease. In the presence of pronounced stenosis its volume is reduced and it is apt to be accelerated and disordered in rhythm.

Percussion.—The cardiac outline is unaltered in many cases, while in the presence of right auricular dilatation the transverse dullness of the heart is extended to the right of the sternum in the third and fourth interspaces.

Auscultation.—The lesion of tricuspid stenosis generates a presystolic murmur with its point of maximum intensity at the tricuspid area over the lower end of the gladiolus, whence it is not transmitted beyond the limits of the lower precordia. In the presence of marked stenosis of the valve the second sounds of the heart are diminished in intensity at the aortic and pulmonary areas at the base of the heart.

Diagnosis.—The detection of a presystolic murmur with its point of maximum intensity in the tricuspid area and frequently a presystolic thrill in the same region, with signs of venous engorgement point to tricuspid stenosis. As tricuspid and mitral stenosis are frequently concomitant lesions, the mitral murmur frequently obscures the tricuspid murmur and leads to an error in diagnosis.

CONGENITAL HEART DISEASE

Clinical Pathology.—As a consequence of arrested development, injury, and diseases of intrauterine life, definite and varied abnormalities arise in the heart and the great vessels springing from it. The majority of these lesions are not incompatible with the continuance of life, and their chief interest to the internist resides in the liability of the imperfect organ to attacks of acquired cardiac disease.

Pulmonary stenosis is the most frequent of the congenital lesions of the heart. The atresia may be complete, the orifice of the valve being closed by a fibrous membrane, or it may exhibit varying grades of patency. In addition to stenosis of the valve, there may be narrowing of the conus arteriosus, or the pulmonary artery may be congenitally narrow beyond the valvular opening.

A further common congenital lesion of the heart is the patent foramen ovale, which normally closes during the first week of extrauterine life, but which may remain partially patent until adult life in 14 per cent of subjects.

The interauricular septum may be absent, resulting in the reptilian heart, or *cor triloculare.* In other instances both interauricular and interventricular septa are absent, the heart consisting of only two chambers, the *cor biloculare.*

In a limited number of cases the ductus arteriosus, which usually closes during the first month of extrauterine life, remains patent to give rise to signs of congenital heart disease.

Congenital lesions of the aortic, mitral, and tricuspid valves are infrequently encountered. Congenital lesions at these valves may consist of supernumerary cusps, a diminution of the number of valvular segments, adhesions between them, or merely a buttonhole slit in a membrane closing the valvular orifice.

In unusual cases the aorta is found to arise from the right ventricle and the pulmonary artery from the left ventricle; while in cases of visceral transposition the heart lies chiefly in the right half of the thorax.

Numerous subjects of congenital heart disease die within the first few days or weeks of extrauterine life, and 50 per cent of the cases succumb during the first five years of life. Among those who survive, many develop pulmonary disease, especially tuberculosis, during early adolescence. These patients are likewise frequently attacked with acute endocarditis during this period of life. On the other hand, defects in the interauricular and interventricular septa as well as patency of the ductus arteriosus frequently are not incompatible with the attainment of the usual span of life.

Physical Signs.—The physical manifestations of congenital heart disease are usually early apparent, the most striking sign being an extreme blueness or cyanosis of the child. However, there are cases in which cyanosis is not a striking feature of the condition. Clubbing of the terminal phalanges is common, the so-called Hippocratic fingers, and dyspnea of some degree is invariably present.

As pulmonary stenosis is the most frequent lesion, there is in most cases a systolic blowing murmur situated to the left of the sternum in the second interspace, attended by signs of hypertrophy of the right ventricle. Patent ductus arteriosus yields a systolic thrill, a rather prolonged systolic murmur which is more distinctly audible in the third left interspace and is not associated with right ventricle hypertrophy. Its duration sometimes extends beyond systole.

In the presence of defects of the interventricular septum Rogers' murmur is commonly audible. This congenital defect of the heart is attended by a loud and rasping murmur with its point of maximum intensity in the fourth left intercostal space between the left sternal and midclavicular lines. The murmur continues throughout systole and diastole, attaining its maximum intensity at the height of systole, and exhibiting a progressive diminution of intensity during ventricular diastole. It is commonly attended by a palpable thrill and a well-defined pulmonary second sound.

The Electrocardiogram.—There is no characteristic electrocardiogram. The *P* wave may be large and the electrical axis may deviate to the right. Notching of the *Q R S* complex and other abnormalities may occur.

Diagnosis.—In many instances a diagnosis of congenital heart disease can be made upon the extreme cyanosis, dyspnea, clubbed fingers, and loud blowing murmurs. It is usually very difficult, however, to determine with any degree of certainty the precise congenital lesion with which we are confronted, as the signs are usually indefinite and confusing, and as frequently two or more congenital anomalies coexist.

In certain cases the murmurs of congenital disease are quite difficult to differentiate from functional murmurs in anemic children; but it should be borne in mind in this connection that functional murmurs are not transmitted and do not produce alterations in the myocardium.

From acquired heart disease, congenital disease is usually differentiated by the fact that it is present from birth, the child from birth having been blue (morbus cæruleus); that the child is usually under two years of age, at which time acquired lesions are seldom in evidence; and that the murmurs are atypical in location and transmission in congenital heart disease.

CHAPTER XLIII

DISEASES OF THE MYOCARDIUM

MYOCARDITIS

While the term *myocarditis* implies an inflammation of the myocardium, in the group of cases which are embraced under this name signs of acute inflammation are seldom in evidence. On the contrary, the myocardial changes are rather degenerative in nature, being represented by cloudy swelling, fatty degeneration, or by a diffuse or insular sclerosis.

As the myocardium through its rich blood supply is intimately exposed to various infections and intoxications, it is not surprising that the heart muscle is damaged to a variable extent during the acute infections and chronic intoxications which frequently arise.

The predominant clinical manifestation of myocarditis is an impairment of the functional efficiency of the heart. This impairment naturally varies with the nature of the lesions and with their distribution. Involvement of the conduction system results in more serious cardiac derangement than does involvement of other portions of the heart.

For the type of myocardial disease which is purely degenerative, Hyman and Parsonnet have suggested the term "myocardosis." Only time will tell whether this term will become a part of our medical vocabulary, but it is important to distinguish in our minds the inflammatory from the noninflammatory diseases of the myocardium.

Clinically, myocarditis is recognized in two forms; namely, as *acute myocarditis,* and as *chronic myocarditis.* While the two forms are frequently encountered as separate entities, in certain instances the pathologic changes and the clinical manifestations do not show a sharp line of demarcation.

ACUTE MYOCARDITIS (ACUTE MYOCARDIAL DEGENERATION)

Clinical Pathology.—Acute myocarditis is prone to develop during the course of acute infectious diseases, notably during typhoid fever, diphtheria, pneumonia, scarlatina, rheumatic fever, influenza, variola, syphilis, osteomyelitis, puerperal sepsis, and general septicemia, diseases which are due to bacterial infection and which are attended by constitutional toxemia. Likewise, in cases of coronary embolism and thrombosis a localized acute myocarditis is always induced.

The pathologic findings in acute myocarditis vary with the exciting cause and with the distribution of the disease. According to the character and distribution of the lesions, two principal forms of the disease are recognized; namely, *acute parenchymatous myocarditis,* and *acute interstitial myocarditis,* which occurs in suppurative and non-suppurative forms.

Acute parenchymatous myocarditis usually has its inception during the febrile stage of one of the acute infections, although in the case of diphtheria and pneumonia it not infrequently has its inception during convalescence. The pathologic changes are largely confined to the muscular elements of the myocardium, assuming the form of cloudy swelling or albuminoid degeneration, although slight interstitial changes are noted in most cases. These changes are usually diffuse, involving an extensive area of the myocardium.

Grossly, the heart is enlarged, and stretching of the auriculoventricular orifices results in relative insufficiency of these valves. The walls of the heart may maintain their normal thickness, but they are usually thinned in the region of the left ventricle. The consistence of the heart is diminished, causing the organ to flatten out upon the autopsy table when it is removed from the body. The color of the myocardium is rather pale, and has been likened to that of parboiled meat or raw fish.

Microscopically, the muscle fibers are swollen, the transverse striation is indistinct, and the fibers contain minute granules of albuminoid degeneration. In more advanced cases fatty degeneration may be noted, although the latter change is infrequent. In still other instances the hyaline degeneration of Zenker is in evidence. The retrogressive changes are particularly pronounced upon the surface of the myocardium; and, in cases of uneven distribution, they impart a mottled appearance to the heart.

Acute interstitial myocarditis is usually noted in connection with acute rheumatic fever, scarlatina, and septicemia. With less frequency it arises during the course of diphtheria, typhoid fever, and other acute infections which are more prone to be complicated by parenchymatous myocarditis.

In the *non-suppurative form* of acute interstitial myocarditis the intermuscular spaces are infiltrated with leucocytes; the coronary capillaries are dilated; minute hemorrhages are frequently noted as a result of rupture of these vessels; and the changes extend secondarily to the muscle fibers, which exhibit swelling and at times areas of vacuolation, nuclear multiplication, and pigmentation. At the same time there is a distinct tendency toward the formation of embryonal connective tissue between the muscle bundles. The changes in this form of the disease usually exhibit a subepicardial distribution, and they involve by predilection the apical area and the base of the heart.

The myocarditis of acute rheumatic fever is largely interstitial in type, although coincident changes are frequently noted in the muscle fibers. In rheumatic myocarditis interstitial nodules, *Aschoff bodies,* are usually to be demonstrated between the muscle bundles. These nodules may be small and spherical, or they may occur in patches or streams with their origin in the interfascicular spaces, whence they penetrate the myocardium for a variable distance. The nodules develop at the expense of the interstitial or periarterial connective tissue, and they invariably occupy a position near one of the branches of the coronary arteries. Microscopically, the bodies consist of a matrix of fibroconnective tissue, containing leucocytes, plasma cells, epithelioid cells, and giant cells containing from five to twelve nuclei. The latter cells are arranged more irregularly than is the case in tuberculous lesions.

A further peculiarity of the lesions attending rheumatic myocarditis is that the lesions have a predilection for the bundle of His, resulting in frequent alterations of the cardiac rhythm and a grave prognosis.

In acute myocarditis of moderate degree the integrity of the myocardium is usually eventually restored. In certain instances, however, death occurs from acute failure of the heart, while in other cases the interstitial inflammation eventuates in chronic interstitial myocarditis.

The *suppurative form* of acute interstitial myocarditis is rare, and bears no etiologic relation to the preceding forms of the disease. The pathologic changes assume one of two forms; namely, localized abscesses which are usually situated in the wall of the left ventricle or the upper part of the interventricular septum, or a more or less extensive purulent infiltration involving the interstitial tissues and secondarily affecting the muscle fibers. Rupture of a solitary abscess into the ventricle is attended by general septicemia, while similar rupture into the pericardium induces purulent pericarditis.

Warthin, Brooks, and Adler have conclusively shown that syphilis is not infrequently attended early in its course by changes in the myocardium which constitute an acute myocarditis. The lesions have their inception about the terminals of the coronary arteries, and consist of interfascicular infiltration with mononuclear cells and spirochetes. The infiltration is followed by fibrosis and sometimes by necrosis.

Embolism or thrombosis of the coronary arteries results in myocarditis in the distribution of the accompanying infarction, which may be nonsuppurative or suppurative according to the nature of the occluding embolus. Resolution may restore the myocardium to a normal state, or thinning may result in cardiac aneurysm or rupture.

In certain cases the pathologic changes are not limited to the myocardium, but involve the cardiac nerves as well. Vincent demonstrated a neuritis of the cardiac plexus induced by diphtheria, and Romberg has noted vasomotor paralysis following pneumonia.

The onset of acute myocarditis is usually insidious during the course of an acute infectious fever, and the disease may for a time remain latent, to subsequently improve or to result in sudden collapse from cardiac insufficiency. When an acute myocarditis attacks a subject of chronic valvular disease, it serves to hasten the fatal issue.

The prognosis of acute myocarditis varies with the nature and the extent of the lesions. Many cases of parenchymatous myocarditis complicating acute infections eventuate in recovery. Frequently, however, the heart remains irritable, and the pulse is accelerated by slight exertion over a prolonged period.

In more severe degrees of acute myocarditis the prognosis is less favorable. Under these circumstances dissolution may occur by progressive cardiac failure, or death may occur abruptly during syncope. Unfavorable signs are a progressive fall in arterial pressure, diminution in the output of urine, embryocardia, and pulsus alternans.

The myocardium is rarely attacked without some involvement of both endocardium and pericardium so that in most cases of cardiac inflammation the term pancarditis is applicable.

Physical Signs.—*Inspection.*—In acute myocarditis the patient presents signs of different stages of cardiac failure, such as pallor, restlessness, dyspnea, and sometimes cyanosis. In cases which are attended by visible cyanosis examination of the lower extremities usually reveals edema. Inspection of the precordia is usually negative, although a displaced and diffuse cardiac impulse is at times to be noted.

Palpation.—Palpation of the precordia reveals a feeble cardiac impulse in many instances, while in other cases the apex-beat is impalpable.

The *pulse* is commonly of small volume, frequently disordered in rhythm, and as a rule is accelerated. Slowing of the pulse may be noted, however, as a result of lesions involving the auriculoventricular bundle.

Percussion.—In acute myocarditis it is seldom possible by percussion to demonstrate any increase in the area of dullness of the heart. Usually the outline which is obtained is of approximately normal dimensions.

Auscultation.—Upon auscultation of the precordia the cardiac sounds have a valvular quality engrafted upon them and they are of relatively high pitch, changes which are particularly noticeable in the first sound at the apex. The pulmonary second sound is commonly accentuated as a result of stasis in the lesser circulation, and a reduplication of the first or second sound is sometimes encountered. Occasionally a soft systolic murmur is audible at the mitral or the tricuspid area.

The Electrocardiogram.—There may be widespread acute myocarditis without electrocardiographic change. On the other hand the electrocardiogram may give many evidences of the myocardial involvement. Common findings are tachycardia, prolonged *P-R* interval, *T* wave changes, notching or slurring of the *Q R S* complex and extrasystoles.

Diagnosis.—The detection of acute myocarditis rests largely upon signs of cardiac insufficiency, frequently masked by the symptoms of an accompanying acute infectious disease. When during the course of such an infection pallor, dyspnea, precordial distress, cyanosis, or signs of collapse supervene, the examiner should bear in mind the possibility of a complicating acute myocarditis. Moreover, in the case of diphtheria and pneumonia the disease may have its inception rather late in convalescence.

CHRONIC MYOCARDITIS (MYOCARDIAL FIBROSIS; CARDIOSCLEROSIS; CHRONIC MYOCARDIAL DEGENERATION)

Clinical Pathology.—Under the term *chronic myocarditis* are grouped a large number of cases of cardiac disease, usually developing in subjects of middle life, which present the common characteristics of sclerosis and hypertrophic dilatation of the heart, with the clinical manifestations of cardiac insufficiency.

Chronic myocarditis occasionally arises as a sequel of acute myocarditis, particularly of the cases induced by coronary thrombosis or embolism; but in the vast majority of cases the disease develops slowly in subjects of aortitis and sclerosis of the coronary arteries. In these cases the nutrition of the myocardium is impaired by an obliterating endarteritis of the coronaries, to which is added in certain instances partial occlusion of the orifices of these vessels by aortitis involving the proximal portion of the aorta. Such a state obtains particularly in cases of aortitis of luetic origin. It is to this type of degenerative heart disease that the term myocardosis mentioned above is applicable.

Chronic valvular disease is almost constantly attended by chronic myocarditis in its latter stages. Similarly, chronic adhesive pericarditis and chronic renal disease which is associated with persistent hypertension in the greater circulation are commonly attended by varying grades of chronic myocardial degeneration.

Chronic pulmonary lesions, such as hypertrophic emphysema, chronic interstitial pneumonia, and bronchial asthma are likewise sometimes complicated by chronic myocarditis, the brunt of the disease falling in these cases upon the right ventricle.

Further etiologic factors of chronic myocarditis comprise focal infections of the tonsils, gums, and gall bladder, the persistent tachycardia of exophthalmic goiter, alcoholism, nicotine, and phosphorus poisoning.

The essential pathologic alterations in chronic myocarditis comprise *fibrosis, fatty change,* and *brown atrophy.* Associated with these lesions are others which arise as logical sequelæ, as noted in subsequent paragraphs.

In the *fibrotic form* of chronic myocarditis the heart is enlarged to a variable degree, and the increase in size involves chiefly the left ventricle, although enlargement of both ventricles and auricular dilatation are not infrequent. In this form of the disease the consistence of the heart is firm; the walls are increased in thickness; and the myocardium presents a reddish-brown color, with here and there white streaks or patches, corresponding to areas of fibrosis. The sites of predilection for the sclerosis are the anterior wall of the left ventricle near the cardiac apex, the interventricular septum, and the papillary muscles. With less frequency the fibrosis involves the right ventricle and the auricles. In certain cases which are induced by chronic intoxications, however, the sclerosis exhibits a rather diffuse distribution.

Microscopically, pathologic changes are found in the interstitial tissues, in the muscle fibers, and in the arteries. There may be wavy bands of fibrous connective tissue running parallel with the muscle bundles, but in advanced cases areas are encountered which are entirely devoid of muscular elements. The muscle fibers are atrophic, and frequently they present hyaline or fatty degeneration. In cases associated with coronary disease, an atheroma or obliterating endarteritis is to be demonstrated in the radicles of these vessels.

The *fatty changes* attending chronic myocarditis comprise *fatty infiltration,* and *fatty degeneration* of the myocardium.

Fatty infiltration of the myocardium is usually noted in subjects of general obesity, although the change is occasionally encountered in the absence of this condition. The heart is invested by a thick layer of adipose tissue, which occupies the furrows and, in addition, penetrates the myocardium, in which it is deposited between and not within the muscular fibers.

Fatty degeneration of the myocardium may be diffuse or circumscribed, and it is attended by a more grave prognosis than is fatty infiltration of the heart. In diffuse fatty degeneration the myocardium is pale, and its consistence is so lessened that the organ frequently flattens out upon the autopsy table when it is removed from the body. In cases of circumscribed fatty degeneration, on the contrary, the color of the myocardium is little altered, save that there are to be noted here and there slightly yellowish patches upon the surface, constituting the "tiger heart" of this condition.

Microscopically, in fatty degeneration of the myocardium the muscle fibers are observed to be loaded with minute fat droplets, which serve to obscure partially the transverse striations of the fibers.

The site of predilection for the pathologic changes in fatty degeneration is the region of the apex of the left ventricle. With less frequency the degeneration involves the interventricular septum, the right ventricle, and the auricles.

Brown atrophy is commonly encountered in the senile heart, which is usually moderately reduced in size. In this condition microscopic brown granules are grouped about the cell nuclei, while the fibers are apt to exhibit coincident fatty changes.

Associated changes in chronic myocarditis comprise aortitis, with or without regurgitation or stenosis at the aortic valve, mitral stenosis or regurgitation, tricuspid regurgitation, brown induration of the lungs, hepatic cirrhosis, and effusion of fluid into the serous cavities and cellular tissues of the body.

The aortitis attending chronic myocarditis is usually characterized by moderate or extreme dilatation of the aorta, which is the seat of atheroma, and the aortic cusps are fibrotic, deformed, and sometimes calcareous. The aortic lesions are often particularly pronounced in the root of the aorta, leading to partial occlusion of the mouths of the coronary arteries, and resulting in malnutrition of the myocardium.

The symptoms of chronic myocarditis are quite variable, and they may long be obscured by symptoms and signs of coexisting morbid states. However, sooner or later signs of cardiac insufficiency, notably dyspnea upon slight exertion or in repose, vertigo, faintness, or indefinite precordial pain, make their appearance. In other instances, on the contrary, the onset of the disease is quite acute, the patient being awakened in the midst of sleep by an attack of so-called cardiac asthma, or as a result of effort in the waking state he may be seized with the violent precordial pain. The last symptom is sometimes occasioned by an acute dilatation of the left ventricle, with coincident stretching of an atheromatous aorta.

The course of chronic myocarditis usually extends over a lengthy period, during which the patient complains of dyspnea upon effort, precordial distress, symptoms of chronic bronchitis, and more or less severe digestive disturbances. Vertigo is frequent, and should always arouse the suspicion of a chronic myocarditis associated with arterial fibrosis. In other instances moderate swelling of the feet and ankles, usually most noticeable at the end of the day, is for a long period the only sign of the disease. In still other cases the onset of the disease is acute, particularly when the myocarditis is associated with aortitis involving the proximal portion of the aorta; and such cases may result in early death in an attack of angina pectoris.

The prognosis of chronic myocarditis is always grave, although the disease may extend over a period of years, with remissions of the acute manifestations of cardiac insufficiency. The ultimate prognosis is influenced largely by the state of the arterial system, and concomitant lesions of the kidneys and liver. Unfavorable symptoms are pronounced dyspnea, pulsus alternans, angina pectoris, and syncope.

Physical Signs.—*Inspection.*—In chronic myocarditis, when the patient is examined in repose, inspection is usually negative, although the apex-beat may be displaced to the left and downward. Dyspnea is readily provoked by moderate exertion.

Palpation.—Palpation of the precordia usually reveals an enfeeblement of the cardiac impulse, which indeed may be impalpable as a result of myocardial insufficiency or from the intervention of the anterior pulmonary borders in hypertrophic emphysema.

The *pulse* frequently exhibits acceleration, and the rhythm is often disturbed. The arrhythmia may assume the form of premature contractions, or it may be due to auricular fibrillation. Pulsus alternans is sometimes to be noted, usually as a transient condition following acute dilatation of the left ventricle and angina pectoris. In certain instances the pulse is slow, as a result of a lesion involving the auriculoventricular bundle, in which event partial heart-block is apt to result in disordered rhythm. The radial artery is frequently palpably thickened. The pulse rate is readily accelerated by moderate exercise, and is usually unduly slow in returning to the former rate.*

Blood pressure readings in chronic myocarditis are variable, depending as they do upon the state of the myocardium and the arterial system, and varying with the presence of left ventricular hypertrophy and dilatation, and with changes in the arteries. In approximately 50 per cent of cases the pressures are above normal. In cases with imperfect compensation, on the contrary, the systolic pressure is usually below normal, with a normal or elevated diastolic pressure, depending upon the state of the peripheral arteries.

In cases of chronic myocarditis associated with failure of the right ventricle, palpation of the upper right quadrant of the abdomen will frequently reveal the inferior margin of the liver at an abnormally low level. Similarly, in such cases it is not uncommon for the lower extremities to pit upon pressure as a result of venous stasis and edema.

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*For further discussion, see chapter on Arrhythmias, page 828.

Percussion.—In the established case of chronic myocarditis percussion usually reveals an increase in the transverse diameter of the heart. In early cases, however, it is frequently impossible to demonstrate any alteration in the cardiac outline.

Auscultation.—In chronic myocarditis the cardiac sounds are frequently muffled or enfeebled, and their intensity, quality, and pitch are estimated with difficulty. It is quite common for the first sound at the apex to be enfeebled or to be replaced by a systolic murmur, which in this instance is due to relative insufficiency of the mitral valve. Not infrequently there is also audible at the aortic area a systolic murmur which is generated in the aortic valves or aorta as a result of atheroma and roughening. Late in the disease a tricuspid systolic murmur is sometimes audible, arising as a result of relative leakage at this valve, and attended by the positive venous pulse and systolic pulsation of the liver.

Auscultation of the pulmonary bases posteriorly will frequently reveal the râles of pulmonary edema late in the course of the disease.

The Electrocardiogram.—Various etiologic factors produce chronic lesions of the myocardium. The term "chronic myocarditis," therefore, may refer to lesions which are quite different from each other in location, in extent and in the effects which they produce on the electrocardiogram.

One of the commonest results of such myocardial lesions is an impairment of conductivity in the a-v bundle. This may conceivably be confined to only a part of the bundle or it may involve many parts. The degree of impairment likewise may vary. There may be partial or complete loss of conductivity in the main stem, in the right or left limb or even in the smaller branches. The effect on the electrocardiogram obviously will depend upon the degree and location of the defect. Only rarely, however, are myocardial lesions confined to a small area. Some part of the conducting system is peculiarly susceptible to injury in myocardial disease therefore, for the subdivisions of this system extend throughout the musculature. This explains the frequent occurrence in "chronic myocarditis" of electrocardiograms indicating defective bundle branch conduction.

In the discussion of the normal electrocardiogram it was stated that the ventricular complex (*Q R S T* group) is a bicardiogram representing effects produced in the right and left ventricles at the same time. Experimental records have been obtained showing only the effects produced in one ventricle. Such a record of the right ventricle is called a dextrocardiogram; that of the left, a levocardiogram. Such experimental records show the following features: (1) The principal deflection of the *Q R S* group in Leads I and III is of increased amplitude and is usually notched or slurred. (2) The duration of the *Q R S* deflection is greater than normal. (3) The initial and final phases of the ventricular complex (i.e., the *Q R S* group and the *T* wave) are oppositely directed in Leads I and III. Human electrocardiograms which show the above features are regarded as indicative of defective bundle branch conduction. Since the defect in different cases may be of different degrees, and since the lesion is rarely confined to a single area as in experimentation, human electrocardiograms which show the classical picture of complete block in only one of the bundle branches are of relatively rare occurrence. Records which indicate more or less impairment of bundle branch conduction, however, are quite common. If the impairment in the right branch appears to predominate, the record is often designated simply as one showing defective conduction in the *right* branch; if the predominant impairment is in the left branch, the record is often referred to simply as indicating a *left* bundle branch defect. In attempting to analyze and interpret human electrocardiograms, however, it cannot be emphasized too strongly that disease rarely performs simple experiments. Human electrocardiograms, therefore, rarely represent isolated lesions, and "typical" records should not be expected in myocardial disease.

In Fig. 315 are reproduced parts of three electrocardiograms each of which shows impairment in bundle branch conduction of high degree. The diagnosis is made from inspection chiefly of Leads I and III. In the first record the *Q R S* complex is slurred in the first lead and notched in the third. Its duration in these leads is about 0.17 of a second. The normal duration of the *Q R S* complex is not more than 0.1 of a second; usually much less. In the electrocardiogram under discussion the direction of the principal deflection of the *Q R S* group is opposite to that of the final (*T* wave) deflection in Leads I and III. The ventricular complex of Lead II in this record (and in the second as well) also shows the above features, although these frequently are not so characteristic in the second lead.

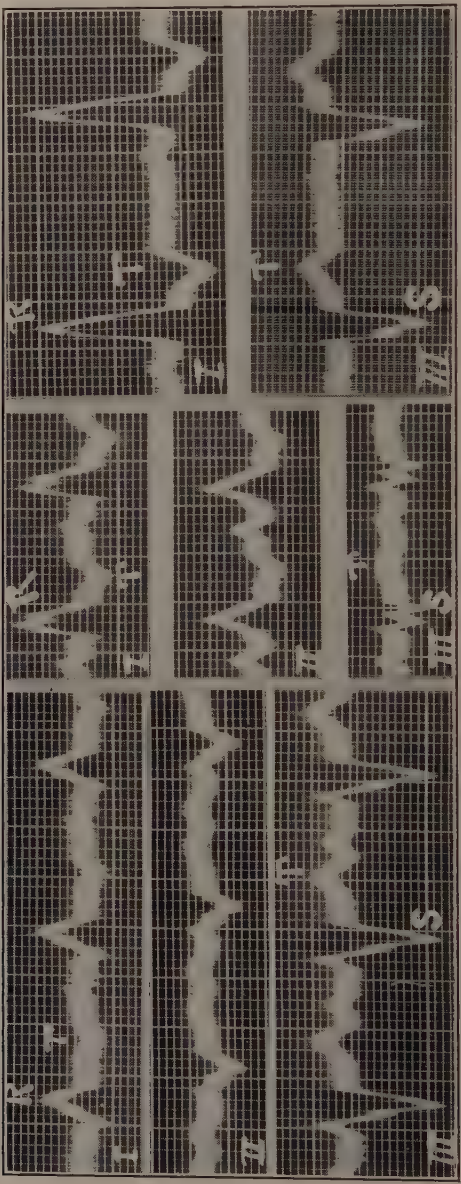

Fig. 315.—Parts of three electrocardiograms, each indicating a high degree of impairment of conductivity in the right limb of the a-v bundle. In the third specimen only Leads I and III are shown. Discussion in the text.

In the second record the *Q R S* complex is slurred and notched in Leads I and III, and is of abnormal duration. The characteristic relationship of *Q R S* to *T* wave obtains. In the third record of Fig. 315 only the first and the third leads are reproduced. These show the characteristic features of defective bundle branch conduction.

In the Fig. 316 are reproduced parts of four other electrocardiograms which also show impairment of bundle branch conduction. Detailed inspection of these records shows that the characteristic features are portrayed in each. A conspicuous difference between the records of Figs. 315 and 316, however, is apparent. In Fig. 315 the chief initial deflection is upwardly deflected in Lead I and downwardly deflected in Lead III, while in Fig. 316 the reverse configuration obtains The ventricular complexes in Fig. 315 present the general configuration of the levocardiogram, those in Fig.

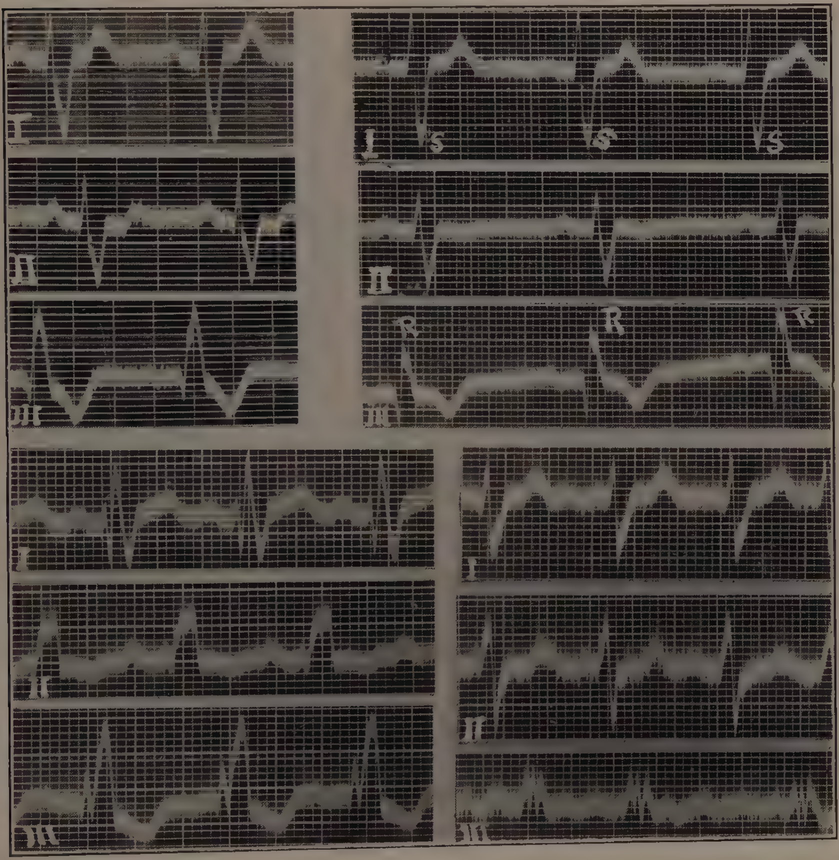

Fig. 316.—Parts of four electrocardiograms, each indicating a high degree of impairment of conductivity in the left limb of the a-v bundle. (The fourth specimen shows also auricular fibrillation.) Discussion in the text.

316 conform to the general configuration of the dextrocardiogram. The records of Fig. 315, therefore, indicate that the left ventricle is activated first, those of Fig. 316 indicating prior activation of the right ventricle. It follows that the chief impairment in conduction in Fig. 315 must be in the right limit of the a-v bundle, the electrocardiograms of this figure representing defective *right* bundle branch conduction. Similarly the records of Fig. 316 indicate defective *left* bundle branch conduction.

In Fig. 315 it will be noted that the general configuration of the ventricular complex of Lead II in the second record corresponds broadly to that of Lead I, while in the first record this configuration corresponds to that of Lead III. Electrocardiograms showing both types occur. A similar variation with regard to Lead II occurs in records from different patients showing defective conduction in the left branch of the bundle. (See Fig. 316.) More commonly in defective bundle branch conduction the configuration of the ventricular complex in Lead II conforms to that shown in Lead III. It should be stated here that electrocardiograms indicative of defective conduction in the right limb of the bundle are of much more frequent occurrence than those which indicate impairment in the left limb. Different theories have been advanced to account for the relative rarity of impairment in the left branch of the bundle. The two most prominent are: (a) The dual blood supply of the left limb renders it less susceptible to injury from *vascular* lesions. (b) The shorter path of the left limb lessens the likelihood of extensive injury from *muscular* lesions.

In Figs. 315 and 316 the degree of impairment in conductivity is considerable, and the diagnosis from inspection of the electrocardiograms would hardly admit of serious doubt. Many other records show abnormalities of the ventricular complex, however, which are not so simply explained. Slurring or notching of the $Q\ R\ S$ complex in the first or second lead, inversion of the T wave in Lead I or in Leads I and II, and axis deviation should be mentioned. Such abnormalities may occur alone or in combination in records which show no prolongation of the $Q\ R\ S$ interval or of the P-R interval. Extended discussion of the meaning of such records would be out of place here. Attention should be directed, however, to a particular type of ventricular complex which is not uncommon. Several examples are shown in Fig. 317. It shows left axis deviation without abnormal prolongation of the $Q\ R\ S$ interval, but at the same time the configuration of the $Q\ R\ S$ to T wave deflections is that of defective conduction in the right limb of the a-v bundle. There is much evidence that such records indicate lesser degrees of conduction defect in the right bundle branch. Theories concerning the exact significance of *T* wave abnormalities should not be considered here. It may be stated, however, that inversion of the *T* wave in Lead I frequently occurs in cases of "chronic myocarditis." If the influence of digitalis can be eliminated, such inversion gives strong indication of myocardial change.

In many instances the exact mechanism responsible for abnormal ventricular complexes is not fully understood. This complex may assume many different forms in different cases of myocardial disease. To include specimen electrocardiograms showing any considerable number of these would be inappropriate. Excellent special works on electrocardiography are available and to these the student is referred for such records.

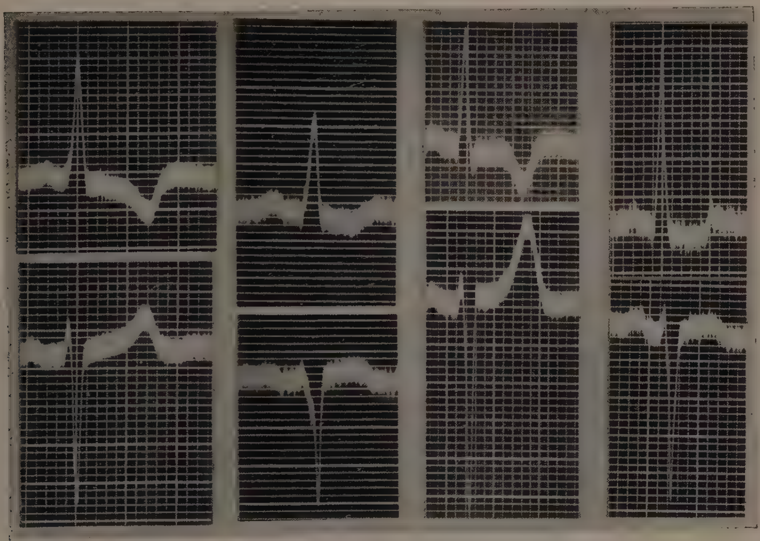

Fig. 317.—Examples of left axis deviation in which the configuration resembles that shown in defective conduction of the right bundle branch; but without abnormal prolongation of the *Q R S* complex. Discussion in the text. (The upper figures are Leads I; the lower figures, Leads III.)

Lesions of the a-v bundle may produce complete or partial a-v block, and electrocardiograms showing such effects might appropriately be discussed here. Since disturbances of rhythm often result from such lesions, however, comment on the accompanying electrocardiograms is deferred for consideration in the chapter on abnormalities of the heart beat.

Radiographs, best taken with the tube six or seven feet from the patient, show the cardiac shadow increased in a transverse direction when the right cavities are dilated, while a ventrical increase is added when the left cavities participate in the enlargement. The aortic shadow is usually increased toward the right, and in cases of luetic origin the changes are most marked in the root of the vessel.

Fluoroscopic examination in late cases shows the ventricular pulsation of small amplitude, indicating a diminution in the functional efficiency of the ventricular musculature.

Diagnosis.—In the presence of symptoms of cardiac insufficiency developing in a subject past middle life, associated with hard peripheral arteries, a disturbance of cardiac rhythm, enfeeblement of the first sound or a systolic murmur at the apex, extension of the cardiac dullness, dyspnea and precordial pain upon moderate exertion, vertigo, and moderate edema of the extremities, chronic myocarditis in its fully established form may be diagnosed.

In other cases the diagnosis may be attended by considerable difficulty. The patient may merely present moderate dyspnea upon exertion, moderate thickening of the palpable arteries and an accentuation of the aortic sound. In such event the examiner will naturally be reminded of the possibility of chronic aortitis with chronic myocarditis arising from malnutrition of the myocardium. Yet other cases run a latent course, with few physical signs save enfeeblement of the cardiac sounds, and transient failures of compensation with signs of cardiac insufficiency.

CARDIAC HYPERTROPHY

Clinical Pathology.—Cardiac hypertrophy, an overgrowth of the musculature of the heart with maintenance of its nutrition, may involve the entire organ, *total hypertrophy,* or it may involve one side of the heart or only certain chambers of the viscus, *partial hypertrophy.*

Total hypertrophy of the heart arises as a sequence of an increased demand for work which is made upon the heart as a whole, usually during intensive athletic training or laborious occupations, chronic adhesive pericarditis, chronic nephritis, and exophthalmic goiter. Likewise, the abuse of alcohol, particularly the ingestion of large amounts of beer, is sometimes attended by general hypertrophy of the heart.

Hypertrophy of the *left ventricle* is induced by arteriosclerosis and chronic nephritis raising the arterial pressure in the greater circulation, by aortic stenosis and congenital narrowing of the aorta, which present an obstacle to the ready emptying of this chamber of the heart, and by aortic regurgitation, as a result of the large volume of blood which the ventricle must expel during systole. Likewise, mitral regurgitation is attended by varying grades of hypertrophy of the left ventricle.

Hypertrophy of the *right ventricle* develops as a result of endocardial and exocardial disease. In the presence of stenotic or regurgitant valvular lesions of the left heart, stasis is induced in the lesser circulation, throwing an increased burden upon the right ventricle, which is met by compensatory hypertrophy of this chamber of the heart. Likewise, in obstructive disease of the lungs, notably hypertrophic emphysema, chronic interstitial pneumonia, and chronic phthisis, an added burden is thrown upon the right heart, which results in hypertrophy of the right ventricle.

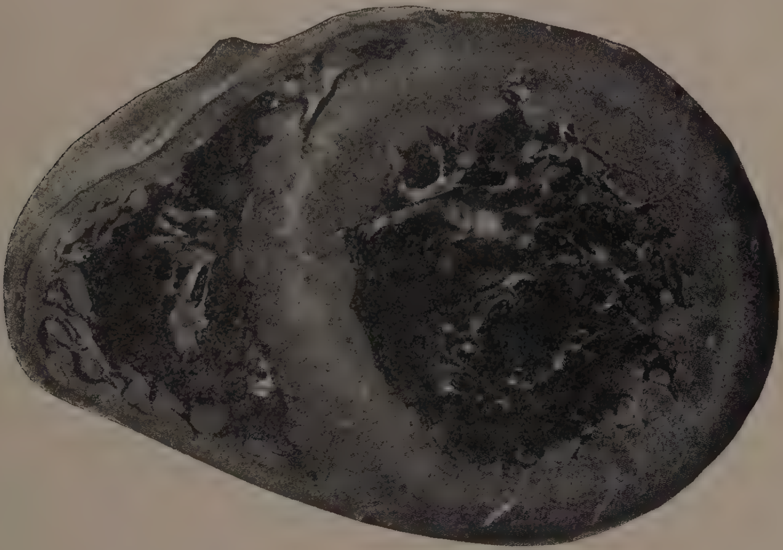

Fig. 318.—Enormous hypertrophy of left ventricle due to prolonged increased peripheral resistance. Note that the whole anterior surface of the heart is occupied by the left ventricle. The right ventricle does not appear to be much affected. (From Warfield.)

Hypertrophy of the *left auricle* is occasioned by stenosis or regurgitation of the mitral valve; whereas, similar hypertrophy of the *right auricle* is induced by tricuspid stenosis or regurgitation, left-sided valvular lesions and obstructive disease of the lungs, all of which raise the arterial pressure in the lesser circulation.

The size and contour of the heart vary with the type of hypertrophy, whether total or partial, and with the underlying cause of the enlargement.

Instead of weighing 280 to 300 grams, in extreme grades of hypertrophy the heart has been found to weigh 1,000 grams. Such hypertrophy is noted especially in the *cor bovimum,* the massive heart attending aortic regurgitation. In more moderate grades of hypertrophy weights varying from 500 to 700 grams are not unusual. Likewise, in hypertrophy of the left ventricle it is not uncommon for the wall to be increased from the normal thickness of 10 to 15 millimeters to 18 to 20 millimeters; and in right ventricular hypertrophy an increase from the normal thickness of 5 to 6 millimeters to 8 to 10 millimeters is frequently noted at autopsy. The auricles, on the contrary, are not capable of very considerable degrees of hypertrophy. As a rule, the thickness of the auricular walls is increased from the normal of 2 to 3 millimeters to 5 to 6 millimeters (Letulle).

In total hypertrophy the heart is increased in size in all diameters, but its contour is little altered. When left ventricular hypertrophy predominates, however, the heart is elongated and the apex is blunt, at the same time being displaced downward and toward the left. In right ventricular hypertrophy the cardiac contour assumes a rather spherical form, with increase in the transverse diameter and very little elongation of the organ. Bilateral ventricular hypertrophy, again, results in moderate elongation, increase in transverse diameter, and blunting of the apical area.

Hypertrophy of the left side of the heart is more frequent than is hypertrophy of the right auricle and ventricle. In relative frequency hypertrophy involves the several chambers of the heart in the following order: left ventricle, right ventricle, left auricle, and right auricle.

Anatomically, two types of cardiac hypertrophy are recognized: *simple hypertrophy,* in which the cardiac wall presents a variable degree of thickening, but in which the cavity is of normal size; and *eccentric hypertrophy,* in which the wall of the heart is increased in thickness, and in which the cavity is increased in size. The latter change in reality constitutes the incipient stage of cardiac dilatation.

In uncomplicated cardiac hypertrophy the myocardium presents a deep red color, and its consistence is firm; but when the hypertrophy is complicated by fibroid or fatty change, the heart presents a more or less mottled appearance, analogous to that which is noted in chronic myocarditis.

Hypertrophy of the heart is a progressive and compensatory change, and it is attended by no subjective symptoms which are referable to the hypertrophy per se. Annoying symptoms such as palpitation, headache, and cutaneous flushing, which are frequently noted in subjects of cardiac hypertrophy, are attributable to coincident changes in the organism.

The duration or maintenance of cardiac hypertrophy depends upon the inherent reserve force of the myocardium, with the lesions which are provocative of the hypertrophy, and with the economic status of the patient. Cardiac hypertrophy may be said to be inadequate and dilatation to have supervened when a cavity of the heart is unable to expel its contents during systole.

Physical Signs.—The physical manifestations of cardiac hypertrophy vary with the type and the distribution of the hypertrophic changes, and with the lesions which have been provocative of the condition.

Left Ventricular Hypertrophy.—Upon *inspection* in left ventricular hypertrophy the cardiac impulse is forcible and heaving, and is displaced downward and toward the left. Moreover, the area of the apex-beat is appreciably extended. Precordial bulging or a widening of the intercostal spaces over the precordia is sometimes to be noted in children and in female subjects with thin chest walls.

Palpation is frequently of service in confirming displacement of the cardiac impulse and in studying its lifting power. In massive hypertrophy the impulse may be encountered in the sixth or seventh interspace, as far outward as the anterior axillary line. In cases attended by hypertrophic emphysema, however, it may be impossible to palpate the apex-beat owing to the intervention of the anterior pulmonary borders between the heart and the chest wall.

The *pulse* is frequently of regular rhythm, but arrhythmia due to premature contractions, partial heart-block, or auricular fibrillation may be noted in late cases of myocarditis with hypertrophy. Likewise, in cases of arteriosclerotic origin the radials may be palpably thickened.

Left ventricular hypertrophy is usually attended by an elevation of the systolic blood pressure. The diastolic pressure, in the absence of valvular disease, may be normal or elevated, depending upon the state of the arterial system. In the immense hypertrophy attending aortic regurgitation the systolic pressure is elevated and the diastolic pressure is unduly low, resulting in a very large pulse pressure.

Percussion reveals an increase in the transverse dullness of the heart toward the left and downward, the left limit of the dullness sometimes approaching the anterior axillary line, and the inferior limit occupying the seventh intercostal space. Percussion of the cardiac outline is more difficult with cardiac hypertrophy than with the normal heart.

Auscultation of the precordia reveals accentuation of the second sound at the aortic area, and the sound is frequently metallic and ringing. In the presence of aortitis a systolic murmur is frequently audible at this area, and in the hypertrophy of aortic regurgitation the second sound is replaced by a diastolic murmur. The first sound as elicited at the apex is frequently accentuated and prolonged as a result of the slow and powerful contraction of the left ventricle. The systolic murmur of relative mitral insufficiency is frequently to be detected in this area.

In certain instances of massive left ventricular hypertrophy a cardiorespiratory murmur is to be elicited along the left border of the heart. As a rule, in such cases the anterior border of the left lung is anchored in front of the ventricle by pleural adhesions.

The Electrocardiogram.—It has been thought that disproportion between the masses of the two ventricles will cause the electrical axis to deviate toward that ventricle whose mass is larger; and axis deviation has sometimes been regarded as strong evidence of hypertrophy. Correlation of electrocardiograms with postmortem findings, however, indicates that there is no characteristic parallel between axis deviation and hypertrophy, except in the case of very large hearts. Other factors, moreover, such as difference in conductivity between the two large branches of the a-v bundle, and position of the heart, are known to cause axis deviation. It would appear, therefore, that axis deviation *per se* affords little evidence of hypertrophy. The explanation is often difficult, but hypertrophy should not be given first consideration in attempting to reach it.

The radiographs show increase of heart shadow, especially downward and to the left.

Right Ventricular Hypertrophy.—Upon *inspection* in hypertrophy of the right ventricle there is frequently to be detected a systolic epigastric pulsation, which is attributable to the powerful thrust of the right ventricle. Similarly, it is at times possible to demonstrate a systolic pulsation along the right sternal border in the sixth and seventh interspaces.

Palpation of the epigastrium reveals the powerful thrust of the hypertrophied right ventricle, which is likewise transmitted to the liver, where it should not be confused with a true expansile pulsation of this organ due to tricuspid regurgitation.

The radial *pulse* presents no characteristics which are attributable to the hypertrophy of the right ventricle.

Percussion.—Cardiac percussion shows that the area of cardiac dullness is extended toward the right, occasionally extending an inch to the right of the sternum.

Auscultation.—The tricuspid first sound is accentuated and somewhat prolonged, and a systolic "safety-valve" leak is occasionally demonstrable when right ventricular failure is imminent. The pulmonic second sound is invariably accentuated and reduplication of the second sound of the heart is frequently encountered.

Left Auricular Hypertrophy.—Hypertrophy of the left auricle is seldom to be ascertained by physical examination. An extension of the cardiac dullness to the left of the sternum in the second and third interspaces is suggestive when found; and, if the hypertrophy is due to mitral stenosis, the presystolic murmur of this lesion is clearly audible as long as left auricular hypertrophy is maintained.

Right Auricular Hypertrophy.—In hypertrophy of the right auricle physical signs are meager, but there is apt to be a demonstrable increase in the area of cardiac dullness to the right of the sternum in the second, third and fourth interspaces. Similarly, presystolic pulsation along the right sternal border in this region is occasionally encountered. Systolic pulsation in the jugular veins, with signs of moderate venous engorgement, point to tricuspid regurgitation with right auricular hypertrophy.

The radiograph shows heart shadow more globular and extending further to the left and right.

Diagnosis.—Cardiac hypertrophy is indicated by the powerful, heaving character of the cardiac impulse; the increase in the area of cardiac dullness in one or more directions; the accentuation of the second sound of the heart; and the hard tense pulse of full volume. However, certain other intracardiac and extracardiac conditions rather closely simulate cardiac hypertrophy and require differentiation.

Fibroid retraction of the left lung may cause a wide impulse, which may during a casual examination suggest cardiac hypertrophy. But the physical signs of cirrhosis of the lung in this instance are quite sufficient to render a differential diagnosis comparatively simple.

Neurotic conditions incident to exophthalmic goiter, and the excessive ingestion of stimulants, as tea, coffee, or tobacco, cause transient vigorous cardiac action, simulating cardiac hypertrophy; but in these conditions the impulse is less diffuse and heaving; the cardiac dullness is not extended; and there are ameliorations and aggravations of the attacks.

In pericardial effusion the increased area of dullness is triangular or pear-shaped with the base directed downward toward the diaphragm; and the heart sounds are enfeebled, and the pulse is of the pulsus paradoxus type.

In cardiac dilatation the heart sounds are feeble, but distinct, and the pulse is feeble and irregular, while cardiac murmurs are to be detected at one or more of the valve areas. At the same time there are signs of general venous stasis incident to right heart failure.

In all obscure cases resort should be had to radiography and fluoroscopy of the heart.

CARDIAC DILATATION

Clinical Pathology.—Dilatation of the heart occurs under three conditions: (1) as a stage in the adaptation of the heart in chronic valvular disease, the *active* or *compensatory dilatation* of Krehl, (2) as *acute dilatation* in subjects of acute or chronic myocarditis, and (3) as *progressive dilatation,* or the *relaxation dilatation* of Sahli.

Active or *compensatory dilatation* represents an early period in the adaptation of the heart in valvular disease, and is a transient change which is followed by compensatory hypertrophy of the chamber of the heart upon which the major burden of the lesion falls. In the same class belong the cases of transient "overfullness" of the heart attending severe physical exertion. Starling has shown that with increased activity of the circulation incident to strenuous exercise the capacity of the heart is increased not only during diastole but also during systole in order to accommodate the relatively larger volume of blood which passes through the viscus per unit of time. Numerous experimental studies seem to indicate that the changes attending such effort do not constitute pathologic dilatation of the heart.

Acute dilatation of the heart ensues when the heart is suddenly called upon to sustain an increased load prior to the development of compensatory hypertrophy. A similar change is induced by more moderate effort when the myocardium is the seat of an acute myocarditis complicating diphtheria, pneumonia, or typhoid fever.

Relaxation or *passive dilatation* of the heart is usually due to atheroma of the coronary arteries in chronic myocarditis, whereby the rapid overgrowth of the heart results in a relative malnutrition of the myocardium. In other instances the dilatation is referable rather to fatty change in the myocardium, resulting in dilatation of one or more chambers as a result of increased endocardiac pressure. Clinically, passive dilatation is recognized when a cavity of the heart is unable to expel its contents adequately during systole.

As in the case of hypertrophy of the heart, so cardiac dilatation may be *total,* involving the entire organ, or *partial,* in which case one or more cavities are dilated.

Total dilatation of the heart, which is noted most frequently as a result of chronic alcoholism or adhesive pericarditis, results in varying degrees of enlargement of the organ, which assumes a roughly globular contour, the so-called "wallet heart." In this form of dilatation the heart is enlarged in all diameters; it is pale in color as a result of coincident fatty and fibroid changes; and its consistence is diminished, the organ flattening out upon the autopsy table when the clots are removed from its cavities.

Partial dilatation may involve one or more cavities of the heart, depending upon the etiologic factors concerned in the change.

Dilatation of the *left ventricle* is noted most frequently in association with late cases of arterial hypertension and in chronic aortitis associated with aortic regurgitation. The dilatation is usually associated with a variable degree of hypertrophy and the secondary myocardial changes incident to chronic myocarditis.

Dilatation of the *right ventricle* attends left-sided valvular lesions which result in insufficiency of the left heart, and obstructive lung diseases, conditions which are attended by elevation of the arterial pressure in the lesser circulation. Owing to the inherent thinness of the right ventricular walls, this cavity undergoes dilatation more readily than does the opposite ventricle.

Dilatation of the *auricles* is usually induced by stenotic or regurgitant lesions at the auriculoventricular valves, the thin auricular walls readily undergoing passive dilatation. In mitral stenosis left auricular dilatation is often noted in the absence of dilatation of the left ventricle, but dilatation of the right auricle is nearly invariably attended by similar enlargement of the right ventricle.

In ventricular dilatation, as a result of yielding of the auriculoventricular rings, the valvular segments are unable to close the abnormally large orifice, with the consequent induction of relative insufficiency. Relative insufficiency of the semilunar valves, on the contrary, is uncommon, as these orifices are seldom enlarged. However, owing to the thinner musculature of the right ventricle the pulmonary valve becomes insufficient in approximately 4 per cent of late cases of mitral stenosis, with the induction of the Graham Steell murmur of relative pulmonary insufficiency.

The symptoms of cardiac dilatation are essentially those of failure of compensation, or cardiac failure, and vary according to the period of the disease and with the portion of the heart which is incompetent. Early decompensation may be manifested by moderate dyspnea upon effort, or a sense of vague precordial distress. Likewise, acute dilatation of the left ventricle incident to sudden exertion in chronic myocardial degeneration and aortitis may be attended by the sudden onset of the syndrome of angina pectoris.

In progressive dilatation of the heart, two general syndromes are presented: namely, *left heart failure*, and *right heart failure.*

In left heart failure, induced by progressive dilatation of the left ventricle and auricle, the symptoms are largely referable to the bronchopulmonary system, occurring as a result of acute or subacute pulmonary congestion. The presenting symptoms are urgent dyspnea, cough with thin frothy expectoration, and ready fatigue upon exertion.

In right heart failure attending progressive failure of the right ventricle and auricle the symptoms are manifested as changes in more remote organs as a result of general venous congestion, such as digestive disturbances, pain over the hepatic region, diminution in the output of urine, and distress relative to accumulation of fluid in the serous cavities and cellular tissues of the body.

Physical Signs.—*Left Ventricular Dilatation.*—Upon *inspection* of the precordia the cardiac impulse is observed to be displaced toward the left and downward, and it is apt to be diffuse and undulatory in type. In some late cases of dilatation of the left ventricle the apex-beat is not to be defined by inspection.

Upon *palpation* the cardiac impulse is defined with difficulty; it possesses little lifting power; and, in late cases, it may be impalpable. A visible diffuse apex-beat which is palpated with difficulty has long been held of great diagnostic significance in dilatation of the left ventricle.

The *pulse* is usually accelerated and exhibits in certain instances disturbances in rhythm due to premature contractions, partial heart-block, or auricular fibrillation.

In left ventricular dilatation the systolic blood pressure is commonly low, and this hypotension is attended by a normal, low, or moderately elevated diastolic pressure, depending upon the presence of normal arteries, aortic regurgitation, or arterial fibrosis.

Upon *percussion* the transverse dullness of the heart is extended toward the left and downward to a degree which is commensurate with the dilatation of the left ventricle.

In late cases daily percussion of the bases posteriorly sometimes serves to show the development of hydrothorax, which is most apt to involve the right side.

Auscultation of the apical area reveals a marked diminution in the muscular element of the first sound of the heart which is brief, of high pitch, and of a valvular quality closely approaching that of the normal second sound of the heart. Similarly the aortic second sound at the right second interspace is diminished in intensity, in striking contrast to the coincident accentuation of the second sound at the pulmonic area. A soft blowing systolic murmur of relative mitral regurgitation is frequently audible at the apex and the aortic area is apt to be the site of a systolic or diastolic murmur due to disease of the aortic valve. Reduplication of the first or second sound of the heart is frequently encountered and the gallop-rhythm of Potain is occasionally to be elicited.

Auscultation of the bases of the lungs posteriorly frequently reveals moist râles due to chronic bronchitis or pulmonary edema, while in the presence of hydrothorax the vesicular murmur is impaired or abolished over the distribution of the serous effusion.

The electrocardiogram may be unchanged or show the features of chronic myocarditis.

The x-ray may show increase of the ventricular density to the left.

Right Ventricular Dilatation.—*Inspection* of the precordia in the presence of right ventricular dilatation usually reveals a very diffuse and undulatory cardiac impulse, which occupies a position along the left sternal border, in the upper epigastrium, or extending to the right of the sternum.

In late cases more or less edematous enlargement of the lower extremities is often to be detected, and moderate cyanosis is not uncommon.

Palpation reveals the slapping character of the contraction of the dilated ventricle, which is in marked contrast to the powerful thrusting movement of the chamber in the presence of right ventricular hypertrophy. Bimanual palpation of the liver at this time will frequently elicit a systolic pulsation of this organ, occurring as the result of tricuspid regurgitation.

Upon *percussion* it is usually possible to demonstrate a minor displacement of the cardiac outline toward the left but not downward; but the principal alteration in the cardiac outline is extension of the dullness far to the right of the sternum, where it encroaches upon the normal pulmonary resonance of Ebstein's cardiohepatic angle.

Percussion of the pulmonary bases usually elicits dullness due to pulmonary edema; and delimitation of the areas of hepatic and splenic dullness reveals an extension of dullness due to chronic passive congestion of these organs.

Auscultation reveals enfeeblement of both sounds of the heart at the apex with diminution in the muscular element of the first sound, which approaches the valvular quality of the normal second sound of the heart. The pulmonic second sound at the base is markedly enfeebled. The soft and blowing murmur of relative tricuspid insufficiency is frequently audible at the tricuspid area. Likewise, in late cases the diastolic murmur of Graham Steell is sometimes audible at the pulmonary area, arising as a result of relative insufficiency of this valve.

Auricular Dilatation.—Dilatation of the left auricle affords few physical manifestations of its presence, owing to the deep location of this chamber of the heart. It may be possible to demonstrate an extension of cardiac dullness to the left in the second left intercostal space, and a visible presystolic impulse is occasionally perceptible in the same region.

Right auricular dilatation is manifested by extension of the area of cardiac dullness to the right in the second and third interspaces and by a visible pulsation in the same area.

In right auricular dilatation and tricuspid regurgitation the venous pulse is of the positive or systolic type.

The electrocardiogram may be unchanged or show the features of chronic myocarditis.

X-ray may show bulging of the right auricle.

Diagnosis.—Advanced grades of cardiac dilatation are recognized with little difficulty by the character of the heart sounds, the increased extent of the area of cardiac dullness, the diffuse and undulatory cardiac impulse, which is displaced from its normal site, and signs of left or right heart failure manifested through adjacent or distant organs.

Early cases are recognized by inadequate response to exercise tests, by blood pressure studies, and by alterations in the radiograph.

Differential Diagnosis.—The enlarged heart of *cardiac hypertrophy* is differentiated from cardiac dilatation by the strong and heaving cardiac impulse, which is clearly visible and is displaced from its normal site, the accentuated second sound of the heart, the full and regular pulse, and the absence of signs of venous engorgement.

The differential points between cardiac dilatation and *pericarditis with effusion* have been described under the latter disease.

Increased dullness to the left of the heart which is due to *consolidation of the left lung* is differentiated by the presence of bronchial breathing and râles over the area of pulmonary disease.

The area of cardiac dullness may also be extended by crowding forward of the heart by the growth of a mediastinal tumor; but these tumors produce pressure symptoms which are more pronounced than any which are produced by enlargement of the heart.

Encysted pleurisy may be confused with cardiac dilatation by broadening the area of dullness around and adjacent to the heart;

but here there is usually the friction sound, and the heart sounds are not altered, and there are no signs of venous engorgement as in the case of cardiac failure.

The presence of hypertrophic emphysema, by the interposition of the anterior borders of the lungs between the heart and chest wall, may effectually mask the cardiac enlargement, and require careful percussion or x-ray examination to bring out the increase in the size of the organ.

CHAPTER XLIV

DISEASES OF THE AORTA

AORTITIS

Clinical Pathology.—Inflammatory changes in the aorta occur in an acute and a chronic form. Acute aortitis has been noted as a complication of acute infections, and certain cases represent syphilis of the aorta in an active stage.

Chronic aortitis results from syphilis, arteriosclerosis, and atheroma involving the aorta. Syphilitic aortitis is particularly prone to involve the ascending aorta and the arch, frequently attacking the aortic valve as well, with the induction of incompetence of this valve of the heart. Aneurysm of the arch is not an infrequent complication.

The arteriosclerotic and atheromatous cases involve the ascending aorta, the arch, and frequently the aortic valves, coronary arteries or the entire arterial system. Aneurysmal dilatation of the aorta and aortic lesions are apt to supervene.

In syphilitic aortitis the predominant change in the heart is commonly an aortic valvulitis, though varying grades of cardiosclerosis may be encountered, as well as lesions of the mitral and tricuspid valves. In aortitis of nonspecific origin the changes in the myocardium are identical with those described under the heads of cardiac hypertrophy and dilatation and under chronic myocarditis.

The clinical manifestations of aortitis vary as the disease is limited to the aorta, or as it involves the aortic valves, the coronary arteries or the general arterial system. In acute aortitis and in cases attended by aneurysm, substernal pain is the rule, whereas in uncomplicated chronic aortitis pain is usually to be attributed to an acute exacerbation of a chronic disease. In the presence of coronary sclerosis typical attacks of angina pectoris frequently occur.

Physical Signs.—*Inspection.*—The signs of aortitis are in general those of arteriosclerosis or aneurysm, depending upon the stage of the disease. In early cases study of the eyegrounds reveals arterial changes.

Palpation.—The peripheral arteries are apt to be palpably thickened, while the pulse is tense and is compressed with distinct effort. The diastolic blood pressure is usually more than 100 mm., with a corresponding elevation of the systolic pressure.

Percussion.—The transverse dullness of the heart is commonly extended in the presence of chronic aortitis, and careful percussion of the area of vascular dullness frequently reveals an extension of dullness to the right or left of the sternum.

Auscultation.—In chronic aortitis the second sound of the heart at the aortic area is accentuated, metallic and ringing, or is obscured by a diastolic murmur. The first sound in this area is frequently attended by a systolic murmur, which is not to be attributed to aortic stenosis, but to roughening or dilatation of the proximal portion of the aorta. The presence of a diastolic aortic murmur points strongly to syphilitic aortitis.

X-ray may reveal changes in the aortic shadow varying from slight widening to the definite bulging characteristic of aneurysm.

Diagnosis.—Signs pointing to arteriosclerosis in a subject past middle age, with precordial distress, a systolic or diastolic aortic murmur, or ringing and metallic second sound at the aortic area, naturally suggest the possibility of aortitis. The x-ray and the specific tests for syphilis reveal the character and cause of the underlying aortic lesion. Similarly, substernal pain speaks strongly in favor of syphilitic aortitis, aneurysm, or an acute exacerbation of chronic aortitis.

ANEURYSM OF THE AORTA

Clinical Pathology.—In the induction of aneurysm of the aorta two factors are interactive in the production of dilatation and partial rupture of the arterial coats; namely, disease of the arterial wall in the form of aortitis, arteriosclerosis and atheroma, and strain exerted upon the vessel wall through arterial hypertension. While disease of the arterial wall may be produced by the toxins of acute infections or through mineral intoxication, syphilis occupies first place in etiologic significance.

Aortic aneurysm is usually a solitary lesion, although multiple dilatations of the vessel have been encountered. The aneurysm may assume the form of a fusiform enlargement of the vessel, or it may occur in the form of a saccular dilatation. The site of solitary aneurysm may be the ascending aorta, the arch, the thoracic aorta or the abdominal aorta.

Saccular aneurysm of the ascending aorta usually springs from the convexity of the vessel and progresses toward the right and forward, reaching the anterior thoracic wall in the second or third intercostal space. Aneurysm of this portion of the aorta displaces the heart downward and toward the left; and the usual termination of the disease is by rupture into the pleural cavity, the pericardium, or rarely into the superior vena cava.

Aneurysm of the aortic arch usually arises from the posterior aspect of the vessel, progressing backward toward the vertebral column, resulting in pressure symptoms referable to compression of the trachea, esophagus, or recurrent laryngeal nerve. Less frequently the aneurysm springs from the anterior wall of the artery, causing erosion of the sternum; and occasionally it has its origin in the concavity of the arch, progressing downward and causing compression of the left bronchus and left recurrent laryngeal nerve.

Aneurysm of the descending aorta usually springs from the posterior aspect of the vessel, between the level of the third and sixth dorsal vertebræ, or just above the diaphragm. In the former case the progressive growth of the aneurysm results in compression of the spinal nerves, left bronchus, or root of the left lung; and rupture may occur into the left bronchus, the trachea, or the esophagus.

The course of the disease is progressive, terminating in rupture of the sac in 75 per cent of the cases. In certain instances the disease remains latent for a prolonged period; but, as in other forms of mediastinal disease, pressure symptoms eventually develop. Dyspnea may arise from compression of the right heart, the trachea, bronchi, pulmonary artery, or vagus nerve. Cough is frequently excessive, arising partially as a result of recurrent laryngeal nerve compression, and partially from interference with the pulmonary circulation and the induction of chronic bronchitis. Dysphagia is apt to arise from compression of the esophagus. Erosion of the sternum or of the bodies of the vertebræ is attended by pain of a dull and boring character; and compression of the vagus nerve is followed by pseudoanginal attacks.

Physical Signs.—*Inspection.*—In the presence of aneurysm of the ascending or transverse arch of the aorta, inspection of the anterior surface of the thorax by oblique illumination is apt to reveal systolic pulsation in the second and third intercostal spaces or in the episternal notch. In the presence of aneurysm of the descending aortic arch a similar pulsation is occasionally noted in the left interscapular region at the level of the scapular spine. In advanced cases of aneurysm of the arch there may be a visible protrusion of the thoracic wall in the superior sternal region, which presents true expansile pulsation during ventricular systole. The cardiac impulse usually occupies a position lower and farther to the left than in the normal subject, as a result of displacement of the heart during the growth of the aneurysm.

Pressure on the sympathetic nerve may produce irregularities of the pupil. Irritation sufficient to stimulate the sympathetic nerve produces constriction, while sufficient pressure to paralyze the sympathetic produces dilatation of the pupil on the affected side.

Palpation.—Palpation with the palm of the hand gently applied to the pulsating area is useful in demonstrating the true expansile pulsation, and in estimating the resistance which is offered by the underlying structures to the impact of the blood stream. Frequently the wall covering the pulsation is quite resistant, while in cases of perforating aneurysm the pulsating area is soft and readily compressible.

The tracheal tug of Oliver may be elicited in many cases of aneurysm of the aortic arch, and should always be sought during the examination.

The *pulse* in aortic aneurysm is of some assistance in estimating the location of the lesion. In the presence of aneurysm of the ascending aorta there is no asymmetry of the two radial pulses. When, however, the aneurysm involves the aortic arch distal to the origin of the innominate artery, the left radial pulse is appreciably delayed, resulting in asymmetry of the radial pulse upon the two sides.

Percussion.—In the presence of extensive aneurysmal dilatation of the ascending aorta there is a demonstrable extension toward the right of the area of vascular dullness in the second and third intercostal spaces. In the case of aneurysm of the transverse arch, there is an extensive area of dullness extending to the right and to the left of the manubrium sterni; and in the presence of aneurysm of the descending arch there is a demonstrable area of dullness in the left interscapular region at the level of the spine of the scapula. Small aneurysmal dilatations of the aorta do not, however, appreciably modify the percussion note of the thorax.

Auscultation.—Auscultation of the thorax overlying an aortic aneurysm is apt to reveal a systolic murmur; but the detection of a murmur is not sufficient ground for a diagnosis of aneurysm. More important than the murmur in a suspected case is the presence of a loud, ringing aortic second sound, which is rarely absent in aortitis or aortic aneurysm. Instead of the accentuated second sound, however, this sound may be replaced by a diastolic murmur. A systolic murmur is frequently appreciable in the carotids in the presence of aneurysm of the arch, but a

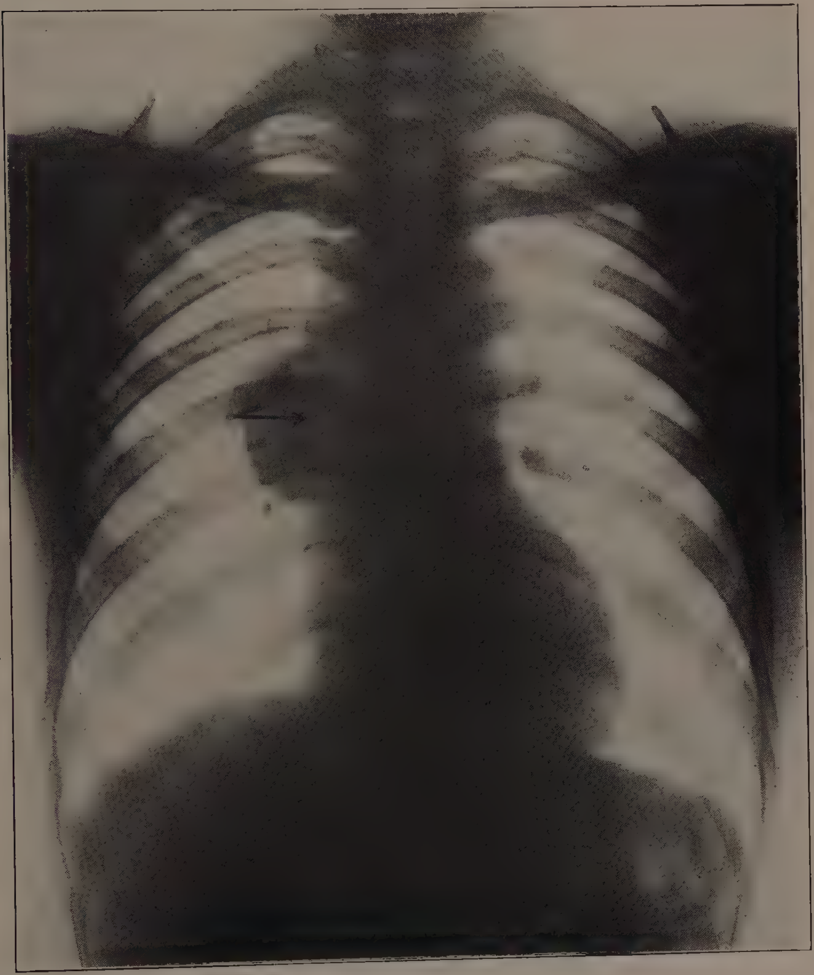

Fig. 319.—Radiograph of aneurysm of the arch of the aorta. Arrow indicates density due to aneurysmal sacculation.

similar transmitted murmur attends stenosis or roughening of the aortic valve in the absence of aneurysm.

The radiograph reveals the presence of a tumor connected with the aortic arch or obliterating it.

Fluoroscopic examination may demonstrate the expansile character of the tumor provided the clot is not so dense as to obscure it.

Diagnosis.—The cardinal signs of aortic aneurysm comprise dullness at the base of the heart, extending to the right or left of the sternum, dullness in the left interscapular region at the level of the scapular spine, pupillary changes, and pressure symptoms, occurring in a subject of chronic aortitis or syphilis. Later in the evolution of the disease boring pain becomes a marked feature of the case, with cough, irregular pupils, cardiac disturbances, asymmetry of the pulses and a visible tumor with true expansile pulsation. Fluoroscopy is of material assistance in establishing the diagnosis and in localizing the site of the lesion.

CHAPTER XLV

DISEASES OF THE CORONARY AND INNOMINATE ARTERIES

THE CORONARY ARTERIES

Coronary Sclerosis

While coronary sclerosis is the basis of myocardial degeneration, there are certain symptoms and signs which point especially to the coronary arteries. Marked coronary sclerosis may exist without evidence of sclerosis of the peripheral arteries. Coronary sclerosis is a factor in the production of some of the cardiac arrhythmias described elsewhere.

Angina Pectoris

The history of an attack of angina pectoris is on the whole typical. The middle-aged are most frequently attacked and men suffer more frequently than women. Syphilis is not as important an etiologic factor as was formerly believed. The attack comes on suddenly with severe vise-like pain, usually over the precordium, extending to the shoulders and often down to the left or right arm. The pain may occur as the result of effort, but frequently occurs when the patient is resting or awakens him from sleep.

It is associated many times with the fear of approaching death. The attacks are usually relieved by two or three drops of a one per cent solution of nitroglycerine dropped upon the tongue, or the inhalation of a small quantity of amyl nitrite. The response to nitroglycerine is a valuable diagnostic sign.

The physical signs aside from evidence of myocardial degeneration are atypical. Sudden death occurs in about thirty per cent of cases. Many cases which were formerly diagnosed angina pectoris have been found to be due to coronary occlusion.

The diagnosis depends upon the history of the attack and in many cases to the relief of the attack following the use of nitroglycerine or amyl nitrite.

Coronary Occlusion

Coronary occlusion has recently become a diagnostic clinical entity. The older conception that coronary occlusion was always fatal has been disproved. Gross has demonstrated that coronary arteries anastomose, thus paving the way for collateral circulation in case of occlusion of branches of a coronary artery.

Clinical Pathology.—Coronary occlusion may be the result of thrombosis, embolism or arteriosclerotic narrowing of a coronary artery at the orifice or along its course. It usually occurs in men passed forty years of age and may or may not follow physical strain. Attacks are frequently noted following a heavy meal.

The symptoms consist of a sudden attack of severe pain, usually located over the precordium, but sometimes referred to the epigastrium or gall bladder region. This pain is less frequently referred to the arms than in angina pectoris. The pain is not relieved by nitroglycerine or amyl nitrite, and even large doses of morphine frequently have little effect.

Profuse sweating is common. Vomiting is common, but rarely relieves the symptoms. Dyspnea and cardiac palpitation are frequently observed.

The attack may partially subside, only to be renewed with increasing severity.

The course of the disease is variable. Occlusion of a large branch of the coronary artery may be followed by sudden death.

The attack may result in marked myocardial weakness, which renders the patient a chronic invalid, or it may subside, leaving little evidence of myocardial damage.

The signs other than physical consist of fever ranging between 99° and 104°, usually not exceeding 101°. Leucocytosis of from 9000 to 16,000. Urinary findings vary from a mild albuminuria to evidence of severe nephritis, said to be the result of absorption of toxic material from the necrotic area.

Physical Signs.—*Inspection.*—During an attack the face may be red, cyanosed, or ashy gray. The patient usually assumes an orthopneic position and grips the cardiac area. The cardiac impulse when visible is faint and often rapid, but not displaced beyond its position before the attack.

Palpation usually reveals a small weak pulse, but arrhythmia does not frequently develop during the attack. At times the pulse may be full and bounding; the apex impulse may be difficult to feel, but is not displaced. A friction rub may be palpated if

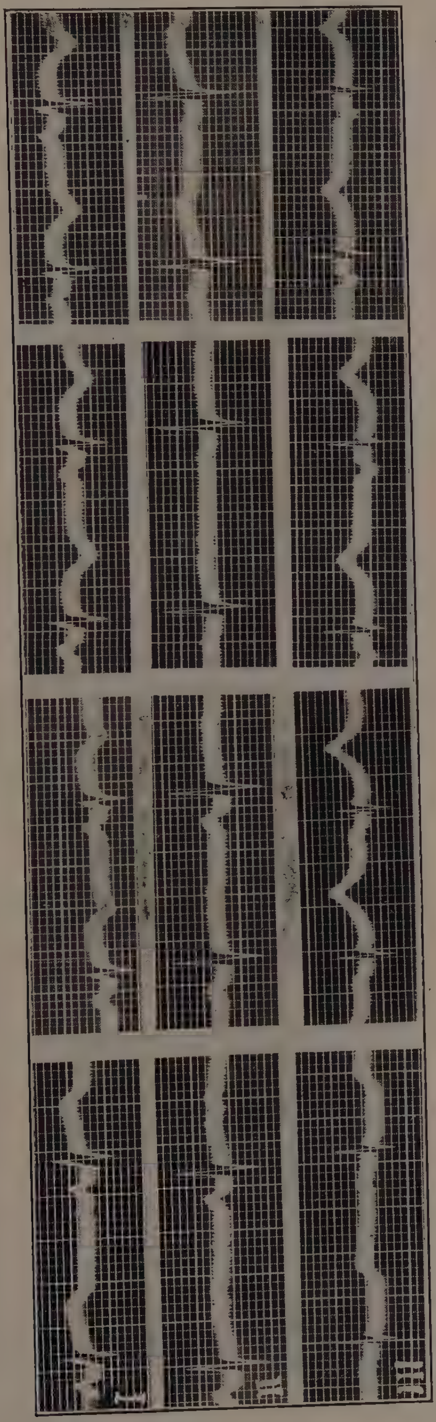

Fig. 320.—Four electrocardiograms from a case of coronary occlusion, made 2, 6, 11, and 16 days following the attack. For description of characteristic features and of successive changes, see text.

the diseased area is superficially placed. This friction rub is, however, often transient, disappearing in a few hours.

The blood pressure falls, the degree of fall depending upon the previous level. The pulse pressure is usually considerably reduced.

Percussion reveals no change in the outline of cardiac dullness.

Auscultation reveals an increase of cardiac rate, often with a shorter, sharper first sound of the heart at the apex. A friction rub may be heard over a small area, but as noted above is often transient. Very rarely is the friction rub followed by evidence of pericardial effusion.

The Electrocardiogram.—The electrocardiogram may be of great value in the diagnosis. Since there is such wide variation in the location and in the extent of injury, there must also be great variation in the abnormalities of the ventricular complex resulting from the muscular insult. This complex, indeed, may on occasion show no abnormality. The abnormal ventricular complex may be of almost any known form. There is one particular type of complex, however, which rarely occurs in any other condition (Fig. 320). It is, therefore, almost pathognomonic, although records of similar form have occasionally been reported in other conditions which produce a measure of *anoxaemia of the heart muscle.* While the importance of such a record, if obtained, can hardly be overestimated, it must be remembered that such a complex is by no means invariably present. Its characteristic feature consists in a branching of the *T* wave at an abnormal level with an obliteration or distortion of the *R-T* interval. This frequently produces a "plateau type" of *T* wave. If records are taken following an attack at intervals of only a few days successive changes will usually be shown.

In Fig. 320 are shown four records from the same case of coronary thrombosis. These were made two, six, eleven and sixteen days following the attack. In the first record the *T* wave of Lead I branches off at a high level, that of Lead III at a low level, its principal deflection being upward in Lead I and downward (or diphasic) in Lead III. The subsequent records show a lowering of the point of origin of the *T* wave with an inversion of its body in Lead I, along with an elevation of the origin and a raising of the body of the wave in Lead III. These changes are rather characteristic but are by no means invariable. The fact that *changes* of any sort are shown over brief time intervals, however, is extremely important. This statement applies in any case of suspected occlusion, whatever may have been the shape of the ventricular complexes at first observed. A circumstance of still greater value perhaps in the diagnosis is this: The demonstration of a *change* in the form of the ventricular complex in a record made shortly after an attack when compared to an available record which was made not long before the attack.

In other electrocardiograms of the general type of those shown in Fig. 320 the *T* wave takes off at a *low* level in Lead I and at a *high* level in Lead III. The characteristic development in such records is for the *T* wave subsequently to be upright in Lead I and inverted in Lead III.

Records of the former type (shown in Fig. 320) are thought by Barnes and Whitten to result from infarction involving the anterior part of the left ventricle, this with the apex, or the apex alone. The left coronary is usually the affected artery. Records of the latter type they think result from infarction which involves the *posterior* part of the left ventricle or this area along with the apex. Usually the thrombus is in the *right* coronary. This type is less common.

A significant feature in many electrocardiograms in cases of coronary occlusion is an obliteration or a shortening of the *R-T* interval. This may be observed in records which previously have shown a high branching of the *T* wave; or it may be an original feature, high branching not having been in evidence at any time.

Diagnosis.—The principal diagnostic points are sudden severe pain, usually over the precordium, but sometimes in the epigastrium or gall bladder region. The pain is not relieved by nitroglycerine. Elevation of temperature, leucocytosis, and albuminuria are usually present.

Precordial friction rub is of great diagnostic value, but is not constant. The electrocardiograph may exhibit the characteristic signs noted above.

Differential Diagnosis.—*Angina Pectoris.*—The pain less frequently extends to the arms, is of longer duration and does not yield to nitroglycerine or amyl nitrite.

The fever and leucocytosis are not found in angina pectoris. Pericordial friction rub when present is a valuable sign of coronary occlusion. The electrocardiogram of coronary occlusion is characteristic in many cases.

Acute Indigestion.—The pain is more severe and sooner or later localizes in the cardiac region. The vomiting in acute indigestion frequently brings relief. Fever and leucocytosis are rare in acute indigestion.

Pericordial friction rub when present is indicative of coronary occlusion. The electrocardiogram will often aid in the diagnosis.

Gallstone Colic.—The pain in coronary thrombosis, while occasionally localized in the gall bladder region, is usually associated with circulatory symptoms, such as dyspnea and cyanosis. Jaundice is frequently present in gallstone colic.

The pericordial friction rub and the electrocardiogram may clinch the diagnosis. The cholecystogram may aid in the diagnosis of gall bladder disease.

Perforating Gastric Ulcer.—There is usually more shock in perforating gastric ulcer, vomiting of blood or bloody stools may be present, and the pain tends to radiate downward in perforating ulcers.

The pericordial friction rub and electrocardiogram suggest coronary occlusion.

THE INNOMINATE ARTERY

Aneurysm of the Innominate Artery

Aneurysm of the innominate artery may occur as an extension from aneurysm of the aortic arch or as a clinical entity. When connected with aneurysm of the aortic arch it appears as an extension of the tumor into the right side of the neck. This tumor usually exhibits an expansile pulsation.

The systolic murmur and diastolic shock may appear as plainly as over the aortic arch.

Primary aneurysm of the innominate artery is comparatively rare. The etiology and clinical pathology, except in cases due to trauma, are the same as in aneurysm of the aorta.

Physical Signs.—*Inspection.*—The tumor appears just above the inner fourth of the right clavicle and may grow to sufficient size to displace the trachea and dislocate or erode the clavicle. The characteristic expansile pulsation is usually present. Edema of the neck is common. The right jugular vein may be markedly distended.

Palpation confirms inspection as to the character of the pulsation. When the aneurysm is confined to the innominate artery the right radial pulse may be weaker and later than the left. The blood pressure may be lower on the right arm.

On percussion the area of dullness is above and below the inner fourth of the clavicle.

Auscultation.—The systolic murmur and diastolic shock are of maximum intensity just above or over the inner fourth of the right clavicle.

Fluoroscopic examination confirms the location and the expansile character of the pulsation.

The diagnosis depends on the location and expansile pulsation of the tumor, together with the radial pulse which is weaker and later on the right side and a lowered blood pressure in the right arm.

CHAPTER XLVI

THE DIAGNOSIS OF ABNORMALITIES OF THE HEART BEAT

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The use of instruments which record graphically certain features of the heart beat has greatly increased our knowledge of the mechanism both of the normal beat and of abnormal beats as well. Along with this better understanding of abnormal rhythms has come an increased ability to diagnose them, so that now a careful clinician hardly needs an electrocardiogram to help him in such diagnosis in the average case. So far as the mechanism itself is concerned, he makes an electrocardiogram only as a confirmatory test in most cases, although instances are not altogether uncommon wherein differential diagnosis is difficult without an electrocardiogram.

In the following pages the clinical signs and symptoms attending the various abnormalities of the heart beat will be enumerated and reference will be made to the characteristic electrocardiogram in each instance. A few records will be reproduced. Chief emphasis, however, will be placed upon the clinical signs, rather than upon the electrocardiogram. The latter is described because it is one of several diagnostic aids and also because, by its graphic portrayal, it helps the student appreciate the mechanism of the disordered beating under discussion.

SINUS ARRHYTHMIA

After an impulse to contraction is elaborated and released at the sino-auricular node, the contraction wave spreads over the auricle and ventricle in turn, i.e., there is an orderly heart beat. Meanwhile another impulse is being elaborated at the sinus, the time interval between successive beats determining the rate. It will be remembered that two opposing factors operate to determine the rate at which the sinus elaborates impulses, the vagus nerves and the sympathetic. Stimulation of the vagus tends to retard the elaboration of impulses, stimulation of the sympathetic (accelerator) tends to enhance the process. Diminution of the tone of the vagus or of the accelerator has an effect opposite to stimulation. The sinus rate at any moment depends upon the degree of vagus and of accelerator tone.

The influence of the vagus predominates, and sinus rate, therefore, is largely a matter of vagus tone at the node. Were it not that a certain degree of vagus tone is almost constantly maintained at the sinus, the latter would elaborate impulses at a rate of 150 or more per minute. But the vagus inhibits automaticity, and because of its restraining influence the sinus normally releases a lesser number of impulses per minute. Change in vagus tone thus results in a change of sinus rate. Many influences cause changes in the degree of vagus tone, and these changes in tone are reflected in slight variations in heart rate from moment to moment. The beats, therefore, are only approximately rhythmic, exact measurements showing slight variations in the time interval between them. The resulting arrhythmia, however, is of such slight extent that, as a usual thing, it cannot be detected by feeling the pulse and examining the apex, but requires instrumental means of demonstration.

In certain individuals, however, these variations in rhythm are of such extent that they may be readily noted. Since the arrhythmia in such cases arises at the sinus area it is called *sinus arrhythmia*. Fig. 321 is an electrocardiogram illustrative of this condition. The varying interval between auricular as well as ventricular complexes is apparent. The beats arise at the normal area, spread over the auricle in normal fashion, are conducted to and involve the ventricle. Each auricular complex is normal. The electrocardiogram shows that the whole heart is involved.

The diagnosis of this condition can usually be made without employing instrumental means. The important point is to demonstrate vagus influence on the arrhythmia. In many cases this demonstration goes on with each respiration; there is a quickening of the pulse with inspiration and a subsequent slowing as expiration takes place. Such a variation of rhythm with normal respiration is common in young children and is observed not infrequently in individuals at the age of puberty. It is by no means uncommon in young adults, but is rarer in older subjects.

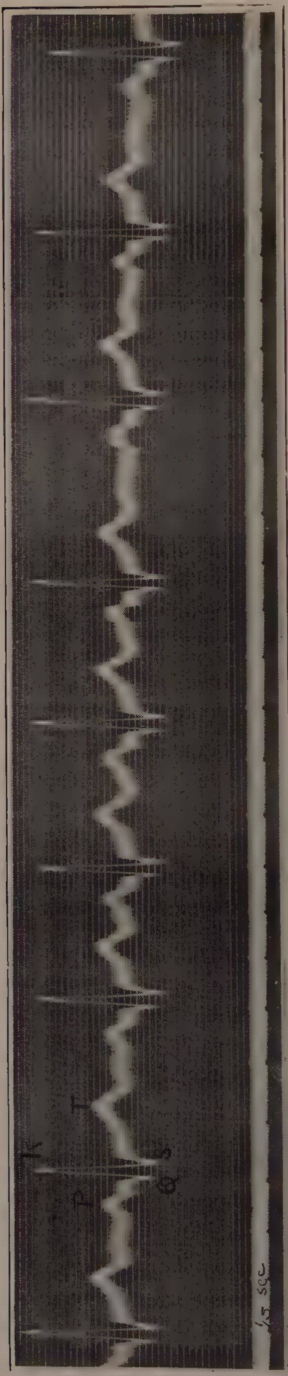

Fig. 321.—Electrocardiogram of sinus arrhythmia. Time in fifths of a second. Note the different time intervals between cycles.

In other examples of sinus arrhythmia no variation synchronous with normal respiration may occur and yet the vagal influence may be demonstrated by having the patient breathe deeply.

There are rare instances of sinus arrhythmia showing irregular variations, in which it is difficult to demonstrate vagus influence or to modify the irregularity. As a rule, sinus arrhythmia is abolished when the rate is made rapid, as by exercise, and is more pronounced when the rate becomes slow again during rest from exercise. The apex shows the same variations in rhythm as the pulse and is of the same rate as the latter. There is no great difference in the loudness or force of individual beats.

Sino-Auricular Block.—In some instances the interval between auricular beats is nearly twice the usual interauricular interval. Such cases are often called *sino-auricular block*. The explanation proposed is that there is an interference to conduction between the sinus node and the main body of the auricle, a sinus impulse failing to reach the auricle and an auricular beat being lost. Observation of many cases, however, shows that the interauricular interval in question bears various relationships to the normal or usual interauricular interval; in some cases being more than twice the standard, in others a little less than twice this length, and in still others considerably less than twice the normal. In other words there is a gradation from "sinus arrhythmia" to "sino-auricular block," and it is better, perhaps, to group all such cases under the former heading.

AURICULO-VENTRICULAR RHYTHM

Reference has been made to the fact that automaticity is highest at the sino-auricular node and that this area consequently becomes the pacemaker. Other parts of heart muscle, however, also possess this property of automaticity, or rhythmicity, and under various circumstances spontaneous beats, singly or in sequence, may originate in areas outside of the sino-auricular node. Such a focus is called an *ectopic* focus, and a beat or a rhythm originating in an ectopic focus is often called an *ectopic beat* or an *ectopic rhythm*. Whether or not ectopic foci are always confined to the specialized tissues need not concern us here. Rhythmicity is highly developed in the auriculo-ventricular node and this node sometimes becomes the pacemaker. It is influenced by both the vagus and sympathetic nerves. The commonest circumstance under which a-v rhythm occurs is depression of the sino-auricu-

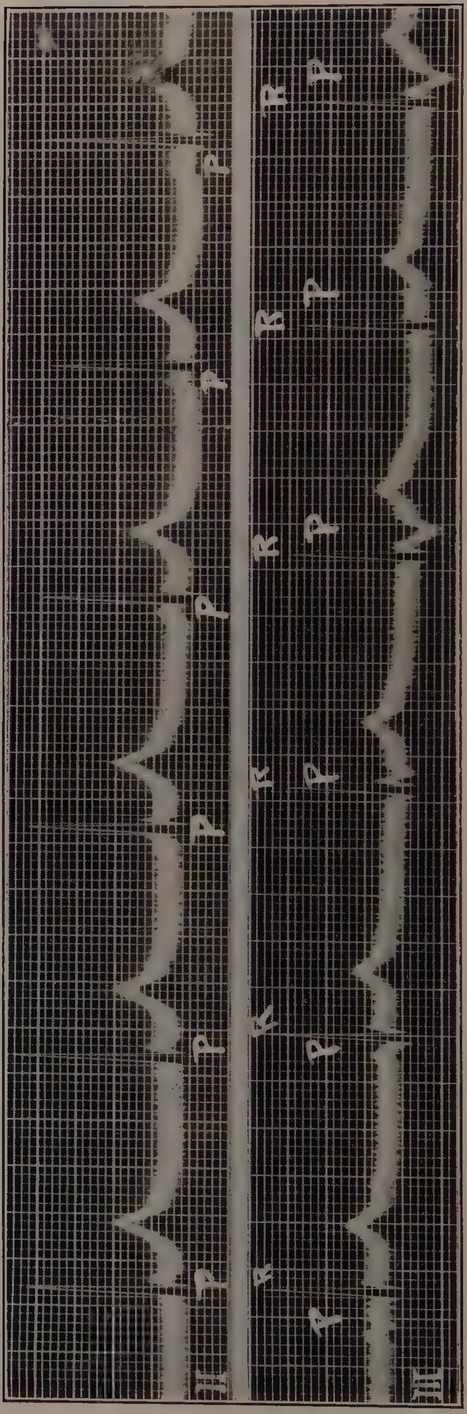
II
P
P
P
P
P
P
III
P
R
P
R
P
R
P
R
P
R
R
P

Fig. 322.—Electrocardiogram of auriculo-ventricular rhythm. For description, see the text.

lar node, the inhibition of the sinus going so far as to reduce its rate below the automatic rate of the a-v node. Auriculo-ventricular rhythm, or "a-v nodal rhythm" may show little irregularity. The diagnosis without a graphic record is usually quite difficult. In many cases the pacemaker shifts frequently from one node to the other, the rates of the two being nearly identical or changing slightly with changing nerve tone.

Fig. 322 is an electrocardiogram in Leads II and III which shows auriculo-ventricular rhythm. In Lead II the auricles and ventricles are beating independently of each other, only parts of the *P* waves being visible because they occur at the same time as the ventricular complexes. It will be noted that the auricular complexes (*P* waves) of the 4th and 6th beats of Lead III are inverted. When these beats were inscribed the a-v node had assumed the rôle of pacemaker and the impulse to contraction entered the auricle from a direction different from that assumed by impulses arising in the sinus node.

Such rhythms are usually abolished by exercise and other procedures which lessen vagus tone, the sinus again assuming the rôle of pacemaker.

PREMATURE CONTRACTIONS

(Extra Systoles)

Interruptions to regular heart action by premature contractions, or so-called "extra systoles," are of common occurrence. Their significance, moreover, is quite varied. It is important, therefore, that the student learn to recognize them and to evaluate them properly. Just what process in the muscle is the immediate cause of premature contractions is unknown, although many precipitating causes are well recognized. Neither the causes nor the mechanism by which they are evoked, however, need occupy us here. It is rather with their clinical manifestations that we are concerned. Careful attention to details will usually enable the student to recognize these signs and to differentiate them from those of other irregularities without employing graphic records.

Reference has been made to the rhythmic origin at the sinus of the impulse to contraction and to its orderly spread over the auricle, through the conducting system, and over the ventricle. The student has been reminded also that an ectopic focus may originate an impulse without waiting for the rhythmic excitation wave. Such an ectopic contraction obviously is premature; it occurs before its rhythmic period. Strictly speaking it should be called a *premature contraction.* The name "extra systole," however, has the force of long usage. Such premature beats may originate from foci located in the auricle, the a-v node, the right ventricle and the left ventricle. More exact localization in these areas is often possible from the electrocardiogram, but without such means the student is sometimes able to differentiate ventricular premature contractions from those which originate above the ventricle, i.e., supraventricular beats.

Before discussing the clinical evidences of premature contractions a few electrocardiographic records will be shown. Reference to these records will aid the student in appreciating the mechanism involved as well as in acquiring diagnostic knowledge of such records themselves. It should be emphasized again, however, that the student should always endeavor to diagnose an abnormal rhythm before making an electrocardiogram. In most cases this can be done.

Fig. 323 shows three electrocardiograms of different patients, each record showing one or more premature auricular contractions. In the first record there are three of these. Following a normal cycle a premature beat in the auricle occurs before the ventricular systole of the preceding cycle is completed; at the time the *T* wave is being inscribed in the electrocardiogram. The *P* wave representing the premature beat is, therefore, superimposed on the *T* wave. The impulse from the premature auricular beat reaches the ventricle and produces a ventricular contraction. Three normal cycles follow, after which there is another premature auricular beat. Two normal cycles follow the second premature contraction before a third premature beat occurs.

In the second record, following five normal beats a premature auricular contraction is recorded, the *P* wave superimposed almost but not exactly on the *T* wave of the preceding complex. Following the next cycle another premature auricular beat occurs. That this second premature contraction of the auricle occurs a little earlier with reference to events in the ventricle, is shown by the fact that the *P* wave is more exactly superimposed on the *T* wave, giving a combined wave which is higher and sharper than in the former instance. The impulse from this second (and earlier) auricular beat reaches the ventricle, therefore, in an

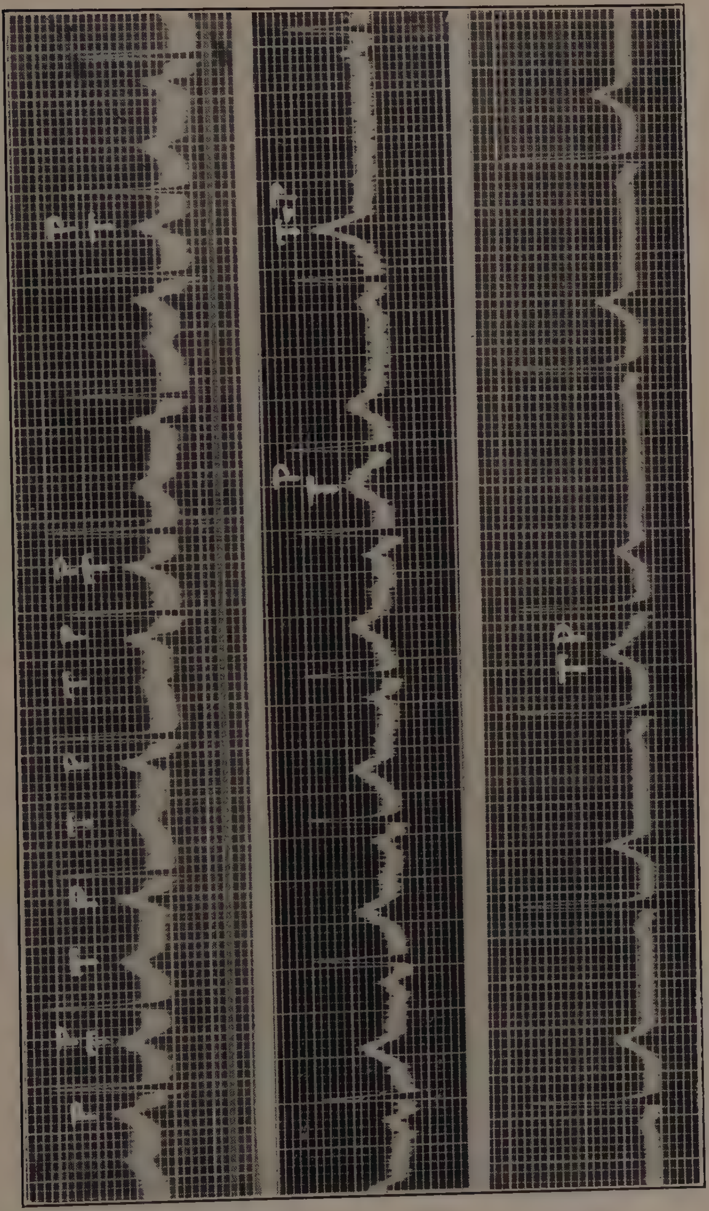

Fig. 323.—Three electrocardiograms showing premature contractions that originate in the auricle. For description, see the text.

earlier phase of its cycle. It arrives during the refractory phase of the ventricle and the latter consequently fails to respond.

In the third record is shown one premature auricular contraction, the *P* wave appearing as a notch on the downstroke of the *T* wave, both giving the appearance of a widened and notched *T* wave. The ventricular complex following the premature auricular beat has essentially the same form as the complexes that follow the normal auricular beats. There are, however, slight variations from the normal in its *T* wave. This is due to incomplete recovery of some part of the conducting tissues or of the ventricle. It is not an unusual occurrence under such circumstances.

It will be observed that in the second and third records the beat of the auricle following the premature beat occurs after an interval which is longer than the normal interauricular interval. In other words, the premature beat is followed by a so-called "compensatory pause." This is also true of the first record, but in this record the difference is slight. In the case of the first record the ectopic focus was probably near the sinus node, or in a different part of the node itself, while in the cases of the second and third records the focus was at some distance from the node.

Under normal conditions the various parts of the ventricle are activated in the same order with the arrival of each successive impulse from the auricle. This sequential activation produces a characteristic form of ventricular complex. If, under abnormal conditions, the impulse reaches certain parts of the ventricle earlier or later than it normally does, this change in the order of activation results in a changed form of ventricular complex. When an ectopic focus *in the ventricle* initiates an impulse it is obvious that those parts of muscle contiguous to the focus are first activated and that parts further away are activated later. An ectopic impulse originating in the right ventricle will spread quickly over the right musculature but will occupy a longer time in traversing the septum before activating the left ventricle. The resulting ventricular complex will differ materially from the normal which, it will be recalled, represents activation in both parts simultaneously. It will assume certain characteristic features of the *dextrocardiogram.* (See page 527.) In like manner an impulse originating in the left ventricle, or a "left ventricular extra systole," will resemble the *levocardiogram.* In both instances the shape of the ventricular complex differs conspicuously from that of complexes produced by impulses which originate *above* the ventricle and enter it in normal manner, activating its various parts in normal sequence. Ventricular complexes of the latter type are designated "ventricular complexes of supraventricular origin," while complexes of the former type are called "ventricular complexes of ventricular origin." It is evident, therefore, that the shape of the ventricular complex is an important point in determining its origin.

In the case of premature contractions which originate in the a-v junctional tissues, the ventricular complex has the general shape assumed by complexes of auricular origin. The exact point of origin of such beats may cause them to pursue a slightly abnormal course, and their ventricular complexes may, therefore, be somewhat unlike the complexes resulting from auricular beats in the same patient. But their general shape will be that of "supraventricular," not that of "ventricular," beats.

Complexes of Ventricular Type.—In Figs. 324 and 325 are shown electrocardiograms in Leads I, II and III which record in each lead premature beats of ventricular origin. Reference to the figures will show that these beats occur before the rhythmic ventricular period and not after a normal *P-R* sequence. They arise independently of auricular influence, and they are, furthermore, of ventricular form. The shape of those which are recorded in Fig. 324 resembles that of the dextrocardiogram. They arise, therefore, in the right ventricle. Those in Fig. 325 have the general shape of the levocardiogram, which indicates that they arise in the left ventricle.

Not all ventricular beats assume such characteristic shapes. Those in Fig. 326 are undoubtedly ventricular in origin. They meet the criteria requisite for this diagnosis, as enumerated above. Those which are shown in the first electrocardiogram of Fig. 326, however, instead of assuming the shape characteristic of either dextrocardiogram or levocardiogram are of the so-called "concordant" type, i.e., their general shape is the same in all leads. More commonly the complexes are of the "discordant" type, i.e., the initial and final deflections are oppositely directed in Leads I and III. (See Figs. 324 and 325.) The shape of the complex in Lead II in discordant electrocardiograms is usually that shown in Lead III (Figs. 324 and 325), although it is not uncommon for it to assume the shape recorded in Lead I (Fig. 326, second record). Lead II, therefore, is not important in differentiating right ventricular beats from left ventricular beats. Complexes of discordant type, because of their resemblance either to the dextrocardiogram or to the levocardiogram, usually offer

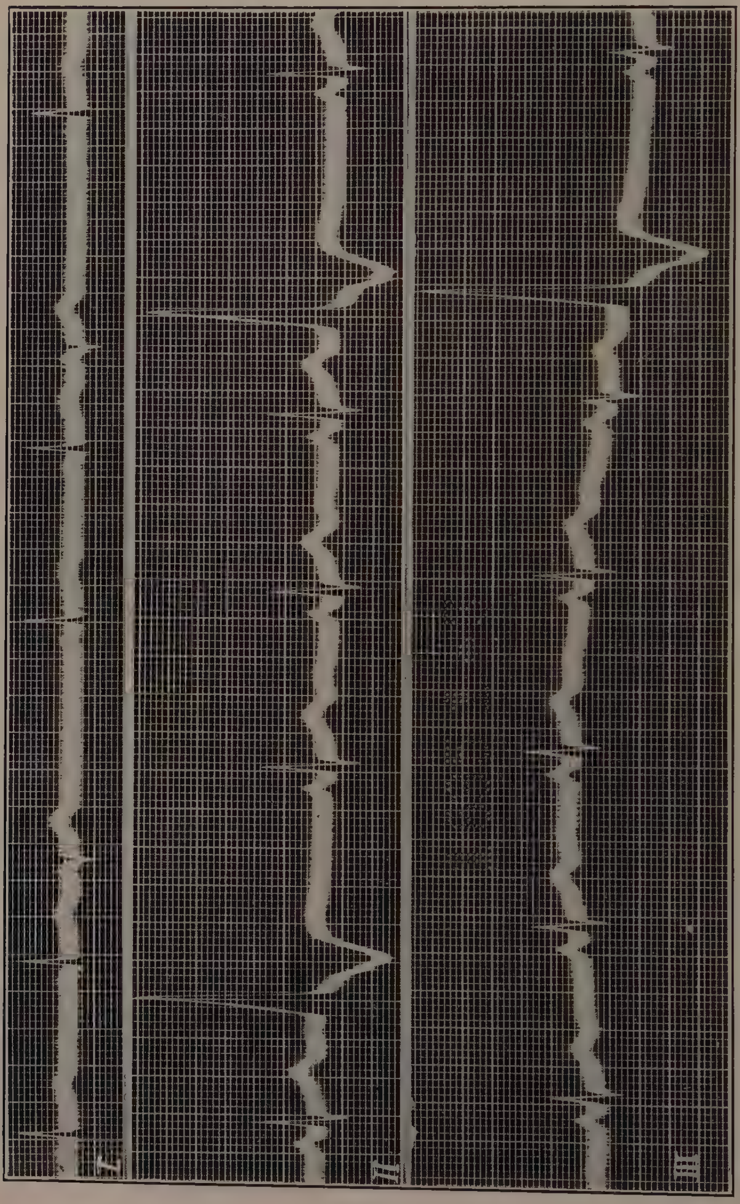

Fig. 324.—An electrocardiogram in Leads I, II and III, showing right ventricular premature contractions. For description, see the text.

little difficulty in determination of origin. In the case of complexes of concordant type that localization suggested by the complex of the third lead is usually preferred.

Complexes of Supraventricular Type.—(a) *Following auricular premature beats:* The ventricular complex which follows an auricular premature beat exhibits a form which is essentially the same as that shown by the ventricular complex which follows the normal auricular beat (see Fig. 323). It is of "supraventricular type." (b) *Following nodal beats:* When an impulse arises in the a-v node it may be conducted upward, causing a contraction of the auricle, and downward, causing a contraction of the ventricle. The ventricular complex, as stated above, is of supraventricular type (Fig. 327). The auricular complex (*P* wave), if any is recorded, usually is of abnormal form. Most authorities say that it is usually inverted. It may be inscribed before, during,

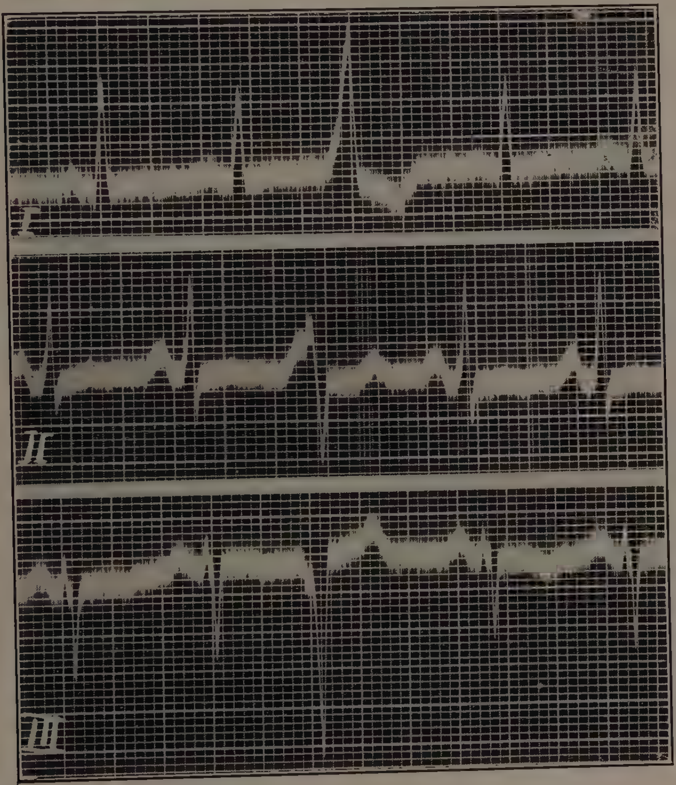

Fig. 325.—An electrocardiogram in Leads I, II and III, showing left ventricular premature contractions. For description, see the text.

or after the *Q R S* complex. It is usually stated that impulses arising in the lower part of the auricle also produce inverted *P* waves. Fig. 328 shows premature beats in all three leads. The ventricular complexes inscribed by these beats have essentially the same shape as that of the complexes which follow the rhythmic auricular waves. The auricular component, however, differs from the normal *P* waves, and the *P-Q* interval is shorter. Whether these beats originate in the lower part of the auricle or in the upper part of the a-v node might be open to question. That they do originate above the ventricle, however, is apparent from the shape of the ventricular component.

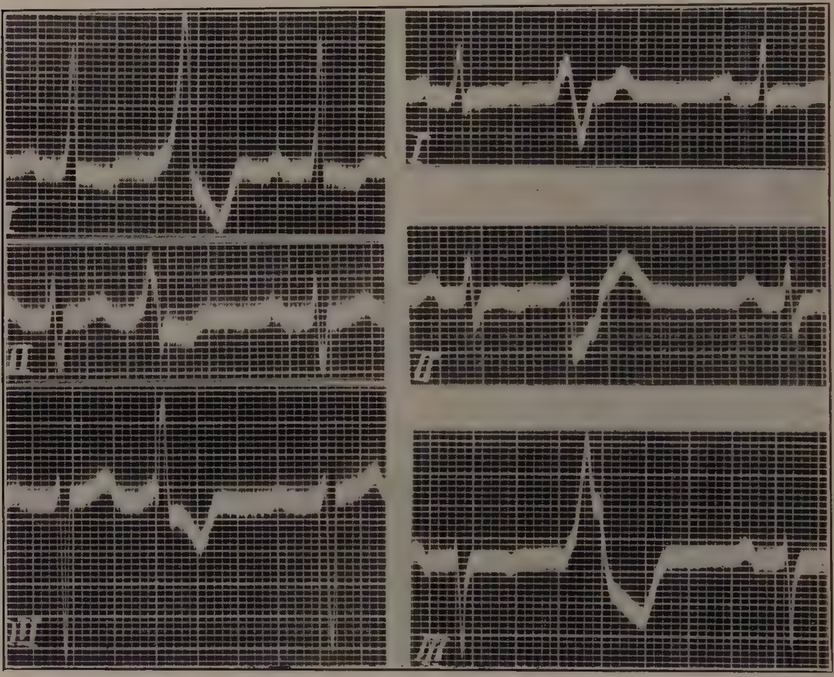

Fig. 326.—Two electrocardiograms in Leads I, II and III, showing ventricular premature contractions. The complexes of those in the first record are of concordant type. The second record shows right ventricular premature contractions, the shape of the complex in Lead II being similar to that in Lead I. Compare with Fig. 324. For further discussion, see the text.

The Compensatory Pause.—It is important for the student to acquire an understanding of the compensatory pause, for upon it depend many of the diagnostic features associated with premature beats. In most cases of ventricular premature beats a rhythmic period of the auricle falls during the time of the premature contraction of the ventricle; the auricular impulse reaches

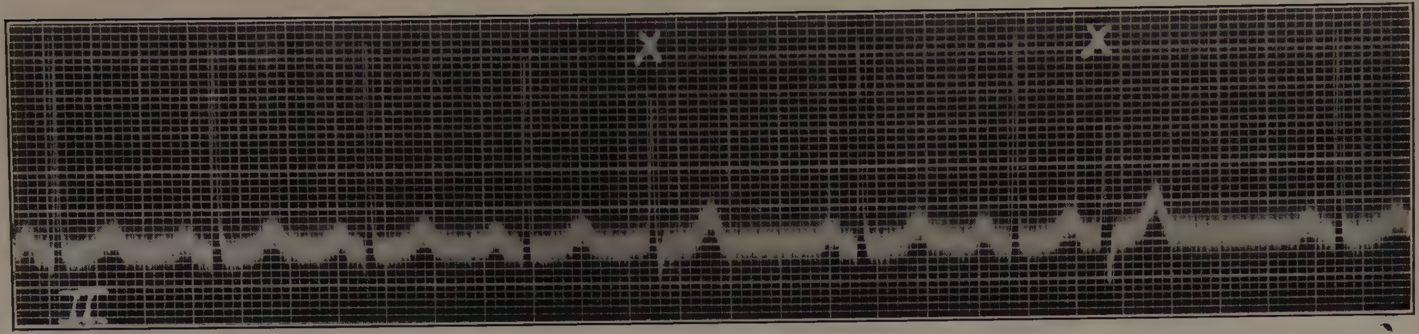

Fig. 327.—An electrocardiogram showing two premature contractions which originate in the a-v junctional tissues. Note the absence of *P* waves and the supraventricular type of complexes.

the ventricle before its refractory phase has passed, and no contraction results. There is a ventricular "pause" which lasts until the next auricular beat reaches the ventricle. This is called the *compensatory pause.* In certain instances in which the auricular rate is slow, the refractory phase of the premature beat may have passed off before the arrival of the auricular impulse. The ventricle will respond to the rhythmic auricular beat in such a case, and no pause will occur. Such premature beats, since they do not disturb the ventricular rhythm, are called "interpolated" premature beats. They are relatively quite rare.

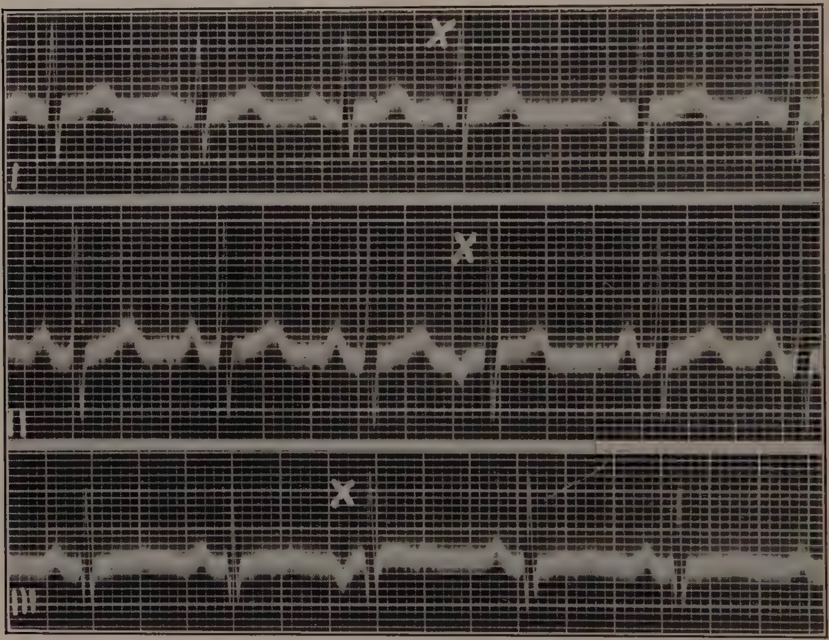

Fig. 328.—An electrocardiogram in Leads I, II and III, showing extra systoles of supraventricular origin. Note the abnormal form of the *P* waves and the supraventricular type of complexes. For discussion, see the text.

It will be observed that the length of the pause following auricular premature beats differs from that which follows ventricular beats. In the case of ventricular beats the length of the pause, being determined by the next sequential auricular beat, is exactly sufficient, when added to the preceding short interval, to equal two interauricular intervals. The pause is truly compensatory. (See Fig. 324.) In the case of auricular beats, however, it is not a true compensatory pause. That is to say, the length of this long interval is not sufficient, when added to the preceding short interval (between normal auricular beat and premature beat of the auricle), to equal two normal interauricular intervals. This is illustrated in the second and third strips of Fig. 323. In the first strip of Fig. 323, as noted above, the premature auricular beat causes little disturbance of auricular rhythm.

Physical Signs of Extra Systoles.—By keeping in mind the mechanism of their occurrence the recognition of premature contractions usually gives no difficulty. The premature contraction, occurring as it does before the ventricle has rested as long as its customary interval, usually gives evidence of its immaturity. The heart sounds produced by it are usually less intense. Its force may be so far diminished that it fails to open the outlet valves, or, opening them, fails to send a pulse wave as far as the radial artery. There may be, then, (a) a small premature radial pulse, followed by an increased interval, or (b) an intermission of the pulse without the premature beat. In the latter case particularly it is important for the student to examine the apex while feeling the pulse, in order to differentiate extra systoles from partial a-v block. If the intermission is occasioned by a premature beat evidences of ventricular activity will be noted at the apex;* while if due to occasional heart block a ventricular beat is missed. If the premature beat is too weak to open the outlet valves, only one (the "first") heart sound will be heard. This may sometimes produce a curious tripling of the heart sounds, the normal sounds being followed closely by the single sound of the extra systole. On listening at the apex, then, one hears the early occurrence of one or both heart sounds followed by a long pause. The finger on the radial artery will detect no pulse wave consequent to the single heart sound, and may find none attending the premature double sound.

Another evidence of immaturity on the part of premature contractions may be noted in connection with their relationship to existing murmurs. A systolic murmur at the apex is usually less intense with the premature beat, or absent altogether. The occurrence with premature beats of existing aortic murmurs will depend upon whether and to what extent the aortic valves are opened by the premature beat.

Premature beats usually disappear if the heart rate is increased to as much as 100. They are occasionally noted at rates higher than 120. They are commonest at slow rates. An extra systole may often be evoked in a susceptible individual by having the subject take a long breath, the premature beat occurring as the heart rate diminishes during expiration.

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*In relatively rare instances the occurrence of an auricular extra systole which fails to excite the ventricle results in an intermission both of the pulse and of the heart sounds (Fig. 323, second strip). The result is the same as if the auricular beat were blocked from any cause.

Symptoms of Extra Systoles.—There may be no subjective sensation accompanying the extra systole. In other cases the subject is intensely disturbed. There is often a "thumping" in the chest. This sensation is usually occasioned by the consciousness of the beat which follows the long interval after the premature beat. It is sometimes produced by the extra systole itself. Many patients notice a feeling as of something rising in the throat or neck. There may be a sort of "all gone" feeling in the chest. The patient may experience a sensation of faintness or of giddiness. Syncope does not occur. In some subjects there is a cough, or a catch in the breath.

While careful examination of apex and pulse usually suffices to establish the character of the arrhythmia when premature contractions are present, the differentiation of auricular from ventricular premature beats is usually not certain without instrumental means. Certain observations, however, suggest their ventricular origin. These are: (a) a pause following the premature beat which appears to be exactly compensatory rather than slightly less than this; (b) a pronounced wave in the neck veins at the time of occurrence of the premature beat. This is accounted for by the simultaneous occurrence of the premature ventricular and the rhythmic auricular beats.

AURICULOVENTRICULAR BLOCK

(Heart Block)

After an impulse has been initiated at the sinus, not more than 0.20 or 0.21 of a second elapses, under normal conditions, before activity begins in the ventricle. Under pathologic conditions a longer time may elapse, or the auricular stimulus may even fail to excite the ventricle. The failure of the ventricle to respond to an auricular stimulus obviously must be due either to the failure of the impulse to reach the ventricle or to its failure to excite the latter after its arrival. For a discussion of theories the student is referred to special texts. It is customary to explain the long a-v intervals and missed ventricular responses in terms of conductivity in the a-v bundle; the length of the *P-R* interval of the electrocardiogram being regarded as the measure of a-v conduction time. This explanation will be employed here.

Fig. 329 is an electrocardiogram showing a delay in a-v conduction. Instead of arriving at the ventricle within the normal time interval each auricular impulse consumes about 0.25 of a second in its passage. The fact that each *P* wave is followed by a ventricular complex, however, shows that all auricular impulses reach the ventricle, even though they are delayed in doing so.

In Fig. 330 a higher degree of conduction defect is exhibited. Near the beginning of the first strip the *P* wave is superimposed upon the *T* wave of the preceding ventricular complex. It is followed by a ventricular complex after about 0.35 of a second. The contraction of the ventricle is delayed so long that by the time it is over, the next auricular beat is due and the second *P* wave also is superimposed on the *T* wave of the preceding complex. The third *P* wave is not followed by a ventricular complex. It fails to elicit a ventricular response. It represents a *blocked* auricular impulse. Interpretation of the second strip will be facilitated by inspection of the fourth, fifth and sixth *P* waves. It will be observed that the sixth *P* wave is not superimposed on a *T* wave. The shape of the *T* wave may be seen where it occurs alone just in front of the sixth *P* wave. The shape of the *P* wave may be seen by inspection of the fifth and sixth *P* waves, no *T* wave being superimposed. Study of the shapes of the isolated *P* and *T* waves shows that the fourth *T* wave is modified by a superimposed *P* wave. This fourth *P* wave represents a blocked auricular impulse. By similar analysis it will be found that the first and second to the last *P* waves of the third strip are blocked, as is also the third to the last in the fourth strip. There was a defect in a-v conduction, in this case, which occasionally resulted in a blocking of the auricular impulse.

The impairment in conductivity may be so great that no auricular impulse reaches the ventricle. In order to understand the mechanism of beating in such a case it may be recalled that ventricular tissues in common with other parts of the heart possess the property of automaticity. Under normal conditions the ventricular pacemaker never has the opportunity to set free impulses to contraction, for its automatic rate is below that of the auricle. Impulses from above continuously discharge the ventricular pacemaker and thus produce ventricular contractions before the ventricular pacemaker itself can initiate an impulse. But when stimuli from above are cut off, the ventricular

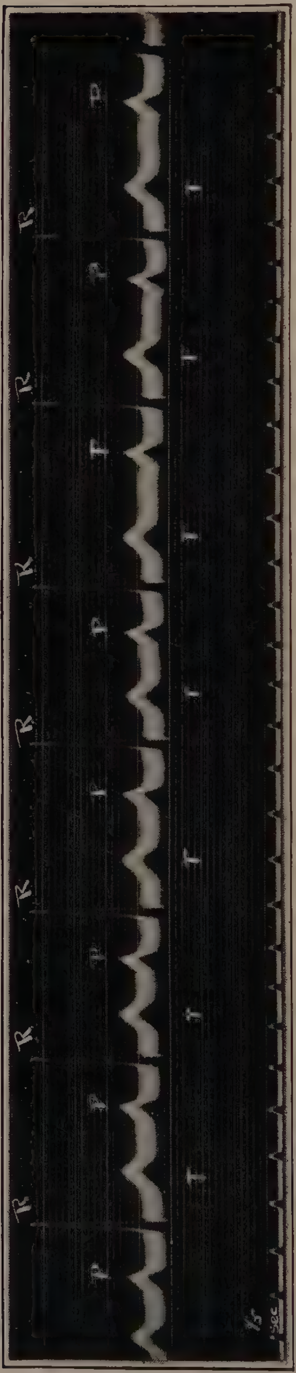

Fig. 329.—Electrocardiogram of delayed conduction. All *P* waves are followed by ventricular complexes, but only after a prolonged time interval. The conduction time averages about 0.25 second.

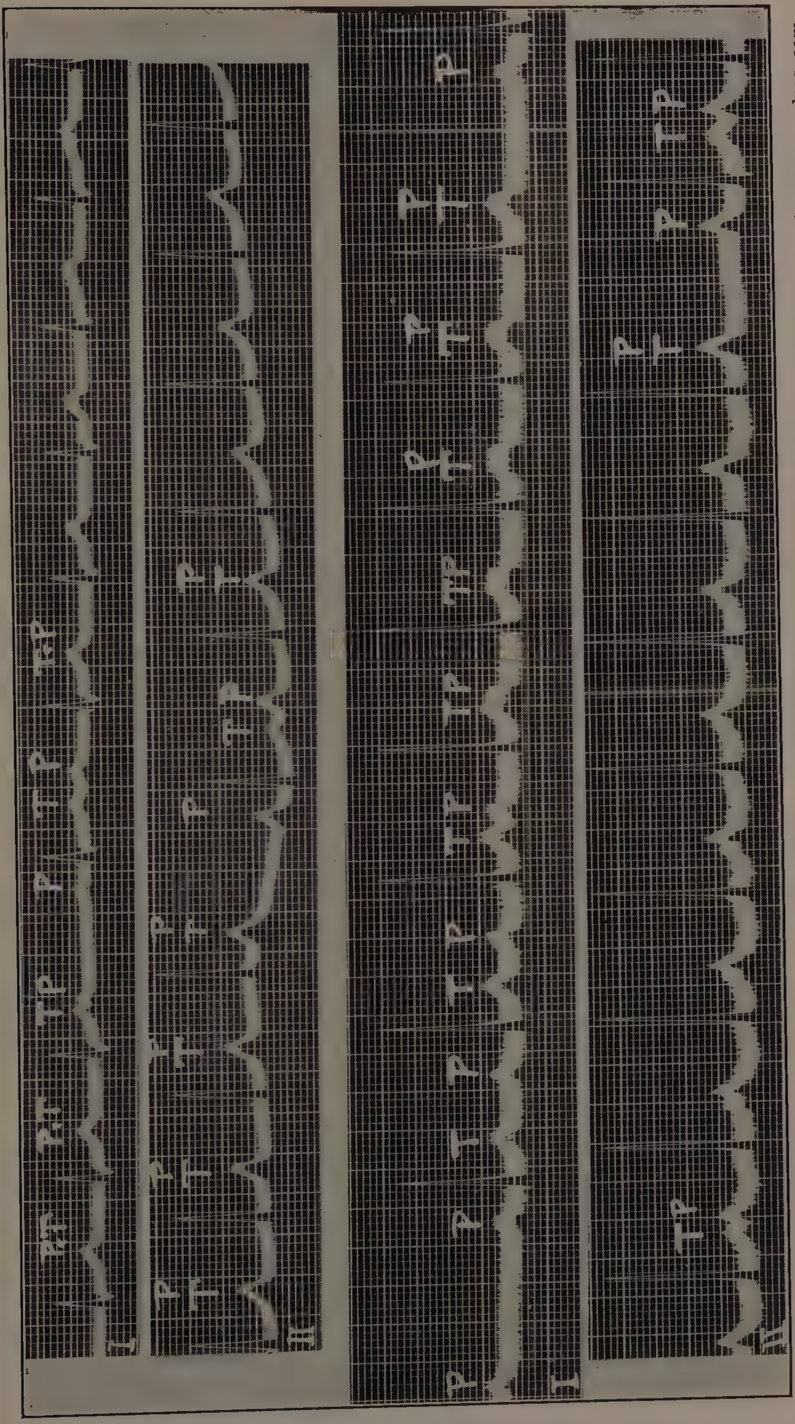

Fig. 330.—Two electrocardiograms in Leads I and II showing partial a-v block. In most instances the *P* wave is more or less completely superimposed on the *T* wave of the preceding complex. In certain parts of the records, however, these waves are separately inscribed and interpretation becomes clear. Further description in the text.

pacemaker, no longer interfered with, begins to assume its physiologic rôle and discharge impulses to contraction. Since its automatic rate is slow the ventricle in complete a-v block characteristically beats slowly. It is often regular, but not infrequently irregular. Its rhythm is necessarily independent of the auricular rhythm. There is *auriculo-ventricular dissociation.*

Fig. 331 is an electrocardiogram in three leads of complete a-v block. Both the complete dissociation between the auricular and ventricular complexes and the slow ventricular rate are apparent.

Signs and Symptoms of Heart Block

Whether the interference to the passage of impulses results in *delayed conduction,* in *partial* (or *occasional*) *a-v* block, or in *complete a-v block (a-v dissociation),* depends upon the degree of interference. This varies under many influences, and this variation may upon occasion result in the occurrence of more than one of these conditions in the same subject within short periods of time. For the sake of clearness, however, each will be considered singly in discussing its clinical manifestations.

Delayed Conduction.—Since each heart beat arises normally and stimulates the ventricle to a normal response, the beats of the latter occur rhythmically, and at a rate determined by the pacemaker. It is impossible, therefore, to establish a diagnosis of delayed conduction without instrumental means of measuring the auriculoventricular interval.

The condition is common in mitral disease. In cases which show the characteristic diastolic murmur and thrill with presystolic accentuation, these signs may be modified by a delay in a-v conduction in a way which strongly suggests, if it does not prove, the delayed conduction. The auricular systole, normally occurring just before ventricular systole (first heart sound) is responsible for the presystolic accentuation of murmur and thrill in mitral stenosis. If, then, the time relationship of auricular systole to ventricular systole is altered, (by a lengthening of the a-v interval), the systole of the auricle will occur not just before ventricular systole (presystolic), but earlier, i. e., in late, middle or early diastole, depending upon the length of the a-v interval. This change in position of the auricular systole by the lengthened a-v interval, will cause a corresponding change in the position in diastole which the accentuated murmur assumes. The murmur, then, may be best heard and the thrill best felt, not just before

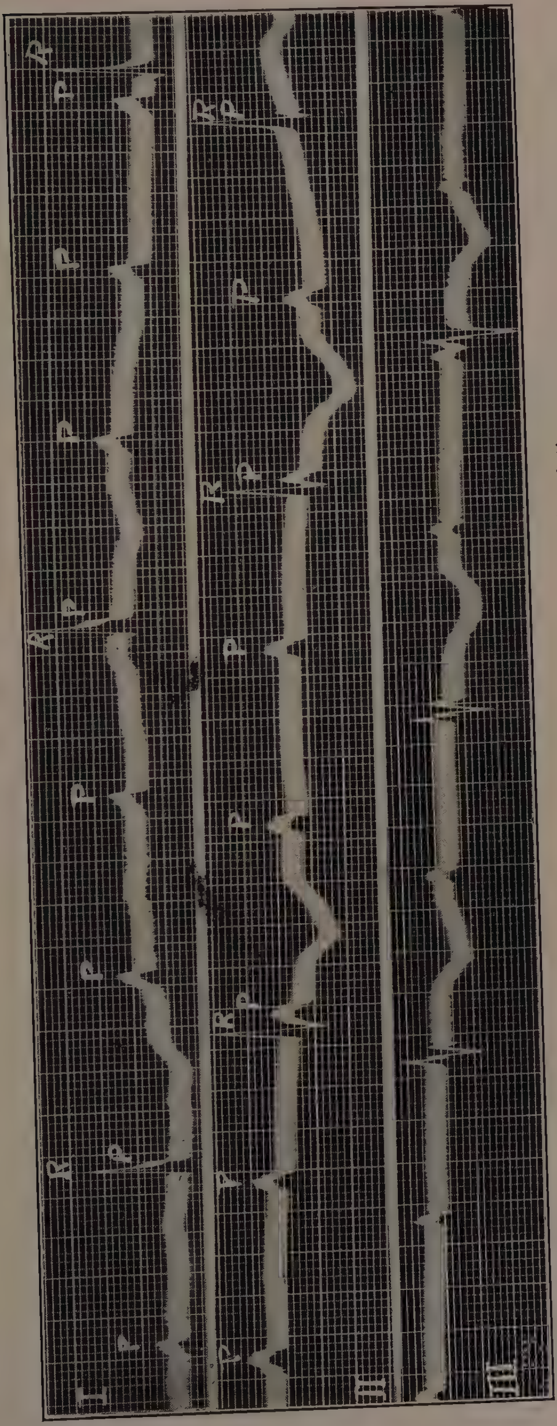
I
II
III
R
P
R
P
R
P
R
P

Fig. 331.—Electrocardiogram of complete a-v dissociation.

ventricular systole but earlier in diastole. Such a relationship of diastolic murmur to ventricular systole in cases of mitral stenosis is always quite suggestive of delayed conduction.

The same altered relationship of auricular systole to systole of the ventricle, separating these contractions by an interval longer than normal, may modify the heart sounds in cases with no valve lesion. The occurrence of auricular systole earlier in diastole than normal may result in its being heard as a "reduplication" of the first heart sound. Its occurrence still earlier in ventricular diastole may cause a "reduplication" of the second heart sound.

Partial Heart Block.—When the ventricle is contracting in response to impulses sent at regular intervals from the auricle, the failure of one of these impulses to reach the ventricle will result in a ventricular pause and an interruption to the rhythm. At that moment no apical thrust against the chest wall occurs, no heart sounds are heard and no pulse is felt. There is a ventricular silence, a true "dropped beat." The pause continues until the next rhythmic auricular impulse reaches the ventricle and causes it to contract. The character of the arrhythmia in a given case will depend upon the frequency and regularity of these dropped beats, i.e., upon the frequency and regularity with which auricular impulses are blocked.

If every second impulse from the auricle fails to reach the ventricle, the rate of the latter will be halved; but, since it is beating in response to alternate rhythmic impulses, no arrhythmia will be produced. Each beat of the ventricle produces a peripheral pulse. Since there is no arrhythmia, this condition, if it persists, may not admit of certain diagnosis without instrumental means. It is rarely persistent, however, and usually shows frequent transitions either to normal rhythm or to other forms of partial block. An abrupt doubling or exact halving of apex rate is quite suggestive. The probability increases if transitions to other forms of partial block occur.

If every third auricular impulse is blocked, the resulting ventricular silences produce a coupled rhythm, a *bigeminy* both of apex and of pulse. Blocking of every fourth auricular stimulus produces a trigeminy of apex and pulse. In like manner different numbers of ventricular beats may be set off by pauses, and the number so marked off may vary within short periods of time.

Not infrequently that heart sound which follows the pause is louder than others. This may give the examiner the impression that he is dealing with extra systoles.

In cases of mitral stenosis, partial heart block may produce striking changes in the murmurs heard at the apex. The blocked auricular systole may produce a murmur similar to the presystolic accentuation heard with other beats. The time relationship of this murmur to the heart sounds will depend upon the relationship of the blocked auricular systole to the next succeeding ventricular systole.

As in the case of sinus arrhythmia and of premature contractions, considerable increase of pulse rate, as by exercise, usually causes the disappearance of partial blocking and produces regularity of the ventricle.

It should be noted that the arrhythmia produced by incomplete heart block is usually modified by changes in conduction time. Particularly is this difference between conduction intervals conspicuous in the case of that interval which immediately succeeds the ventricular intermission. The interval frequently lengthens with successive cycles. These changes in the a-v interval are illustrated in Fig. 330. The irregular occurrence of the block along with these different a-v intervals may produce a complex type of arrhythmia quite difficult to differentiate from certain cases of premature contractions without a graphic record. It may simulate auricular fibrillation.

There may be no subjective sensations. In other cases the patient is aware of the irregularity, the symptoms resembling those associated with premature contractions.

Differential Diagnosis.—It is to be noted that premature contractions may occur with such frequency or regularity as to produce a pulse not unlike that of partial block. Thus a begeminal pulse may be produced by the regular occurrence, after every second normal beat, of a premature beat so weak that no radial response is produced by it. In the case of such premature beats, however, evidences of ventricular activity are present at the apex during the pulse pause, while in the case of partial block *the ventricle is silent during the intermission.* A bigeminal pulse may be produced by premature contractions in another way. After each normal beat a premature beat of sufficient strength to reach the wrist may occur. The pauses following these premature beats will set them off in pairs. In such a case, however, the second pulse is weaker, and the heart sounds produced by the premature beat also are weaker.

The differentiation of occasional ventricular intermissions from sinus arrhythmia is to be accomplished by noting the effect of respiration upon the latter.

Complete Heart Block.—Since the ventricle, deprived of auricular stimuli, establishes its own rhythm in this condition, its beats usually occur slowly. The rate is usually under 40, though not infrequently higher rates are observed. Both heart sounds are heard with each systole, and each contraction produces a radial pulse.

Under favorable conditions the examiner may be able to hear the sounds produced by the contractions of the auricle. These, when audible, are heard occurring rhythmically and with no constant relationship to the regular heart sounds produced by the ventricle. Their rate, derived, as it is, from the pacemaker, is more rapid than that of the ventricle, and is about that of the normal heart.

Even in cases in which the rhythmic auricular sounds cannot be demonstrated, their occurrence may modify the sounds produced by the ventricle. When a contraction of the auricle occurs simultaneously with the first or second heart sound, it may produce a noticeable increase in intensity of the sound which takes place at the same time with it. Its occurrence just before the first, or immediately following the second sound may produce a "reduplication" of the sound near which it occurs.

The rhythmic auricular systoles produce small jugular pulsations which may be visible in the neck during ventricular diastole. When one of these waves coincides with that produced by ventricular systole, i. e., when auricular and ventricular systoles fall together, a visible accentuation of venous pulse may be noted.

The patient may not be conscious of the slow forceful beating of the heart so long as it remains regular. In the severer grades particularly irregularities are likely to occur and there may be periods of excessive ventricular slowing. This slow ventricular beating accounts for the grave symptoms which may accompany complete a-v block. *Adams-Stokes syndrome* is produced by a period of ventricular standstill whose duration is sufficient to cause temporary cerebral anaemia. Dizziness, faintness, syncope may occur, the severity of the symptoms depending upon the length of time the brain is unsupplied with blood. Convulsive seizures, and not uncommonly death, may result.

AURICULAR FLUTTER

The auricular pacemaker normally initiates contractions which vary in rate within rather wide limits. Even with a constant rate, changing vagus tone produces small variations also in the lengths of the interauricular intervals.

Under certain abnormal conditions, however, auricular movements take place at a rate far above normal limits and with great regularity. These contractions are not under the influences that are usually exercised at the pacemaker. The rate is constant and the degree of rhythmicity is extremely high. To this condition of rapid and regular contractions of auricular musculature the term *auricular flutter* has been applied.

There is much evidence to indicate that these movements of the auricle arise in a circus movement that has become established in a natural ring of auricular musculature. Whatever the cause, the condition is characterized by rhythmic auricular movements of high rate. The rate is usually not far from 300 per minute, although it sometimes is considerably above or below that figure. It is remarkably constant. In many instances auricular flutter persists for long periods of time, although transient attacks are by no means uncommon.

There is almost always a certain amount of auriculoventricular block, the most common form consisting of 2:1 block. The rate of the ventricle, therefore, is lower than that of the auricle, and may be one-half, one-fourth or some other fraction of the auricular rate. There may be irregular ventricular response.

Fig. 332 shows parts of two electrocardiograms of auricular flutter. The top strip is a record in Lead II. The bottom strip shows parts of a record in Leads I, II, III. In the top record one sees ventricular complexes evenly spaced, the *T* waves being downwardly deflected. Between each pair of ventricular complexes there is the same succession of waves. The latter represent auricular activity. The trough of an auricular wave coincides with each *R* wave and is partly obscured by the latter. Another auricular wave is distorted by each *T* wave. Keeping these facts in mind one sees that there are four auricular waves to each ventricular complex. The record shows "4:1" flutter. The lower strip of Fig. 332 shows "2:1" flutter in Leads I, II and III.

Fig. 333 is an electrocardiogram of auricular flutter in all leads which shows frequent irregularity of the ventricle.

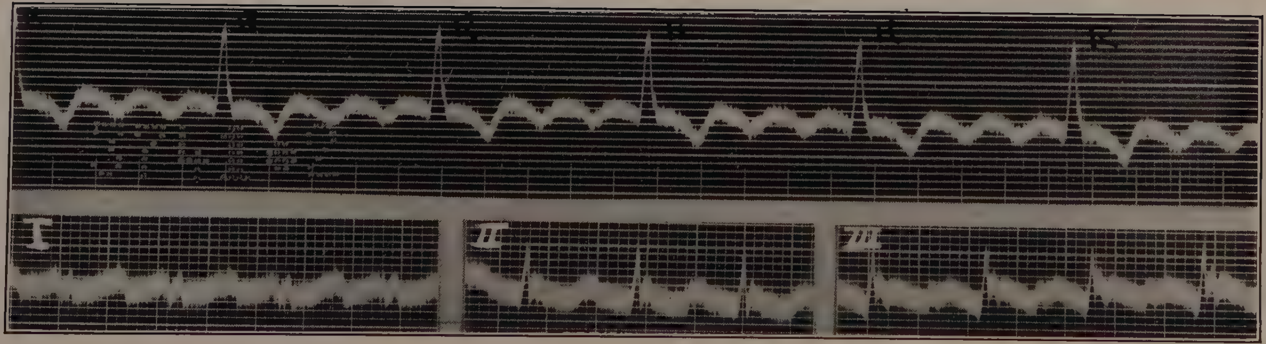

Fig. 332.—Two electrocardiograms of auricular flutter. The top strip is a record in Lead II. In it four auricular waves to one ventricular complex are inscribed. The *R* and *T* waves distort the auricular waves that are synchronous with them. The second strip is a record of 2:1 flutter in all three leads.

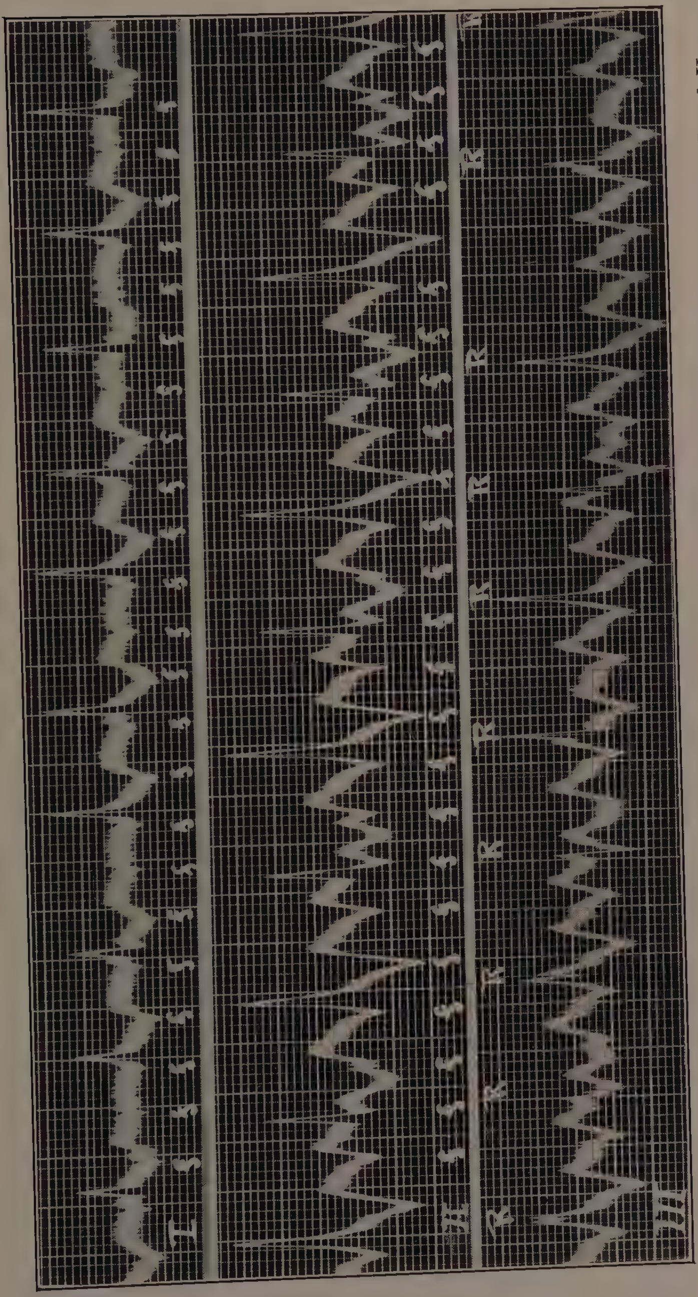

Fig. 333.—An electrocardiogram in Leads I, II and III of auricular flutter. The auricular waves in Leads I and II are marked *f*. Note that they are smallest in Lead I. Note also the irregularity of the ventricular complexes in Leads I and III.

Signs and Symptoms.—Cases may be divided roughly into two general groups: In one group there are attacks of palpitation, the duration varying from a few moments to several hours or days. There may be consciousness of the unusual heart action even though there be no irregularity. Irregularity in some cases is a prominent feature. If the ventricular rate is fast, as is usually the case in this group, the patient may experience a sense of weakness or of faintness.

The second group is composed mainly of individuals who show evidences of arterial sclerosis. The ventricular action is regular and there are no subjective sensations which direct attention to the abnormal cardiac mechanism *per se.* There may be 2:1, 3:1 or 4:1 block, and the ventricular rate, therefore, may be about 150, 100 or 75. A normal heart rate and the absence of special symptoms and of irregularity may cause the condition to go undiagnosed unless certain suggestive evidences are kept in mind. *The persistence of the same heart rate* in an elderly individual is suggestive, particularly so if the rate is high.

In any case there may be a suggestive quality to the heart sounds. The sounds are often somewhat faint or there may be a suggestion of embryocardia. The flutter waves may be visible in the neck veins.

The pulse in the majority of cases is regular but irregularity is not infrequent since the blocking of auricular impulses does not always take place with regularity. Such arrhythmias are difficult to diagnose without graphic curves. The ventricular beats, taking place as they do in response to rhythmic impulses from the auricle, occur at intervals which may be anticipated by taking account of the rhythm. But this is difficult to accomplish with accuracy without instrumental means. Exercise, however, of small amount may cause the disappearance of this arrhythmia, and produce the unvarying rate which is characteristic of auricular flutter. An important diagnostic procedure is the application of pressure over the carotid sheath. In most but not in all cases, vagus pressure so applied will produce an abrupt slowing of the ventricle for at least a few cycles. This is due to an increase in the degree of block with increase in vagus tone.

Occasionally each auricular contraction may produce a beat of the ventricle for short periods; there being, for a time, no blocking of the auricular impulses. The subject of such attacks usually loses consciousness until a resumption of partial block slows the ventricular rate.

AURICULAR FIBRILLATION

The normal systole of the auricle is an orderly and coordinated affair. The contraction spreads from pacemaker to a-v node, involving successive portions of auricular musculature in sequence; and by producing, for an interval, synchronous shortening of all muscle parts, expels blood from the chamber.

Under certain pathologic conditions this orderly sequence of events does not take place. The auricular muscle quivers and twitches in various places in extreme disorder. No concerted action, no synchronous shortening of all muscle parts, occurs. There is no systole of the whole chamber, and blood is not expelled. The auricular activity appears to consist of rapid fibrillary twitchings in all parts of the musculature. This disordered auricular action is called *auricular fibrillation.* It has a close association clinically with auricular flutter, and the explanation of this uncoordinated activity which at present appears most reasonable is that it, too, is produced by circus movement in auricular muscle. Instead of a ring of auricular muscle undergoing simple contractions, it is supposed that depressed conduction and, in addition, local variations in conduction have so broken up the waves from the ring movements that many complex waves find their way through the auricular musculature. Whatever the cause, the result is disordered action of the auricle which affects ventricular activity in a characteristic way.

The ventricle, no longer receiving rhythmic stimuli of constant type from the auricle, now receives stimuli which vary in strength as well as in periodicity. It responds to such stimuli as are of threshold strength provided the time of their arrival does not coincide with its refractory phase. It is estimated that the total number of auricular waves in auricular fibrillation averages about 400 or 450 per minute. Since there are many potentially effective auricular stimuli per minute, the rate of the ventricle obviously depends upon the efficiency of the a-v conducting system. If conduction is unimpaired the ventricular rate is rapid, in response to the great number of auricular impulses that arrive in a short period of time. Instead of being allowed a period of rest, for recuperation and preparation for the next beat, the ventricle must respond as best it may to the various impulses that crowd down upon it. Some find the ventricle little prepared to contract. Others arrive to find it better able to respond. The result is that the beats of the ventricle vary markedly in strength and in rhythm.

If, on the other hand, there is a considerable degree of impairment of a-v conduction, many of the smaller impulses fail to reach the ventricle. Its rate may not be increased. With slow rates the force of its beats is greater and there may be little variation either in the force or in the periodicity of its contractions. In such cases the presence of auricular fibrillation is not infrequently overlooked.

Fig. 334 shows two electrocardiograms from cases of auricular fibrillation. No waves characteristic of normal auricular activity (*P*) occur. In place of these there are numerous irregular waves, caused by the fibrillary movements of the auricular musculature. The ventricular complexes are easily recognized. The *R* waves are quite distinct, but the *T* waves are more or less distorted by the fibrillary waves which are superimposed on them. The ventricular complexes occur with extreme irregularity. There are no compensatory pauses. A short interventricular interval may be followed by another short interval or by a long one. Several short or long intervals may occur in sequence. There is no predominant rhythm.

Physical Signs and Symptoms.—The characteristics of ventricular action, as recorded by the electrocardiograph, are noted upon clinical examination. The pulse is absolutely irregular; now fast, now slow, now showing an intermingling of rapid and slow beats.

Not only is the rhythm irregular, but there is marked irregularity also in the force of the beats. In general there is a direct relationship between the length of the interventricular interval and the force of the following beat. But in many cases there are frequent exceptions, long pauses being followed by weak beats or short pauses by strong beats.

On listening at the apex the gross irregularity of ventricular action is usually more apparent than upon examination of the pulse. Loud and faint sounds occur with no regularity or relationship to each other. Many of the weaker beats may not open the aortic valves and in consequence only the first heart sound will be heard with them. With these and, it may be, with other somewhat stronger beats, no radial pulse occurs. In this way the apex rate may be considerably higher than the pulse rate. In such a case there is said to be a *pulse deficit.* These signs are more pronounced when the ventricular rate is fast, as it always is

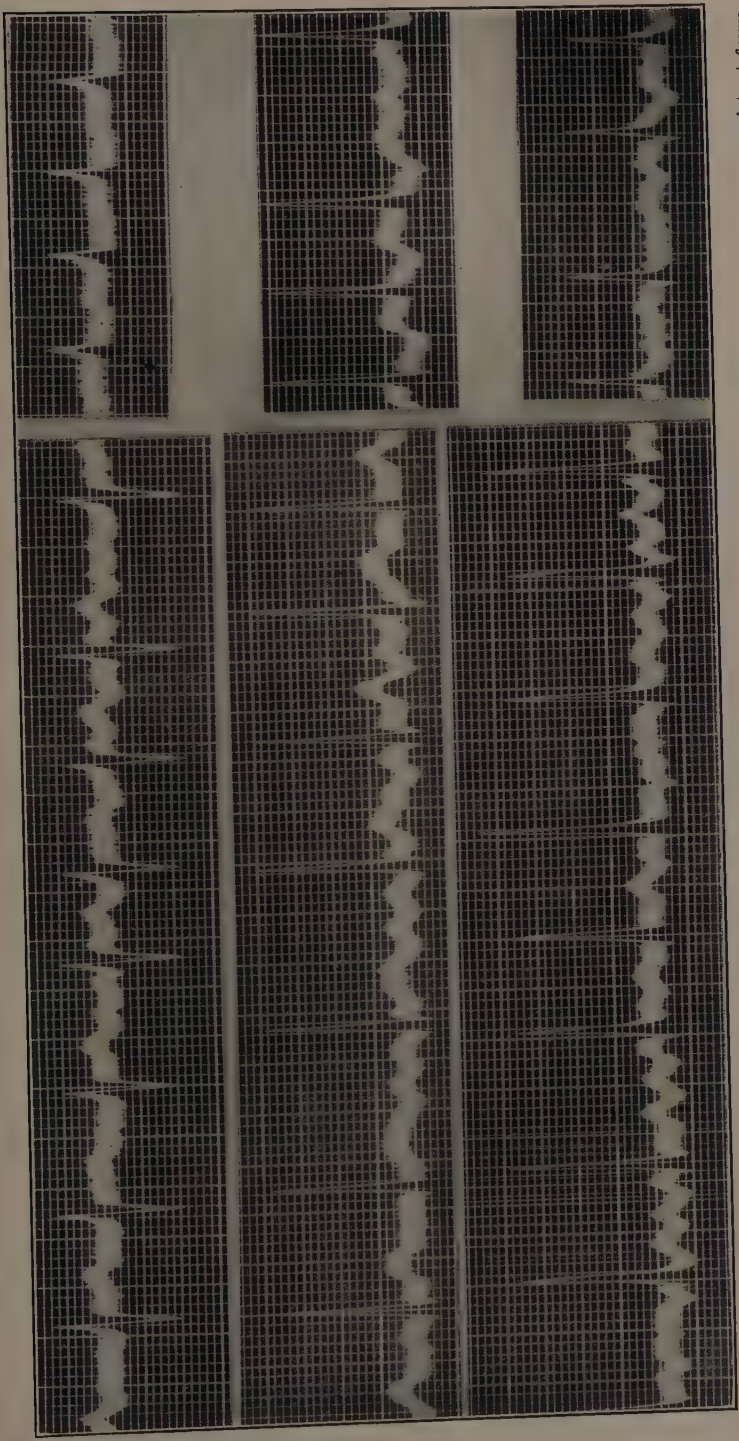

Fig. 334.—Two electrocardiograms in Leads I, II and III of auricular fibrillation. Note that the auricular waves have no consistent form and that they occur irregularly. Note also the total irregularity of the ventricular complexes. In the second lead of the first record the *T* waves are upright; in the second lead of the second record they are inverted. On the *T* waves are superimposed various shapes of auricular waves.

unless partial blocking of auricular impulses is present. When the rate is quite slow with a considerable degree of a-v blocking, there may be little variation in the force and rhythm of the ventricular beats and there may be little or no pulse deficit. The diagnosis in such cases may be quite difficult without a graphic record. When the ventricular rate is fast the variation in the force of individual beats may be strikingly demonstrated by applying the sleeve of a sphygmomanometer and counting the arterial pulsations at various levels of pressure.

Exercise may cause increase in the ventricular rate. With an increase in rate, however, the characteristics of ventricular action become more pronounced, while in other types of irregular action of the ventricle, an increase in rate tends to lessen the arrhythmia. Arrhythmia due to any cause other than auricular fibrillation is uncommon with ventricular rates as high as 120.

Auricular fibrillation is frequently associated with mitral disease and causes a modification of the thrill and murmur which characterize stenosis of the mitral valve. Since no systole of the auricle takes place, the sharp presystolic accentuation of the murmur and thrill do not occur. The time of greatest difference between the pressure in auricle and ventricle, in cases of fibrillation of the auricle, is at the instant when the ventricle is empty and relaxing; i. e., at the beginning of diastole. The flow of blood through the stenosed auriculoventricular opening is fastest at that moment, therefore, and the signs which it produces are most in evidence at that time. The thrill and murmur, then, are most intense early instead of late in diastole.

In cases which exhibit an aortic diastolic murmur, the latter, of course, does not occur with those weak beats which do not open the aortic valves.

Auricular systole is not an essential part of the mechanics of the circulation. Many patients with auricular fibrillation experience no symptoms of deficient circulation. The failure of the auricle to contract in auricular fibrillation impairs the circulation only in so far as this failure exercises a deleterious effect upon the ventricle. Symptoms of heart failure, therefore, never accompany auricular fibrillation *per se*. They may supervene as a result of the fibrillation, however, and thus indirectly be due to the latter. The ventricle, except in cases with impaired a-v conduction, is constantly being goaded by rapidly arriving stimuli. It is deprived of its regular periods of rest. This overwork taxes its reserve, and, in cases in which the latter has already been drawn upon, may be enough to induce decompensation or to add further symptoms if decompensation is already present.

The patient may be conscious of the irregularity. This is particularly common when the rate is fast and the irregularity great. In many patients the tumultuous heart action is quite distressing. Not uncommonly there are brief attacks of fibrillation, and these, of course, produce only brief symptoms, but as a rule the condition is of long standing, once it has become established.

PAROXYSMAL TACHYCARDIA

Under normal conditions the rate of the heart is determined by the degree of tone maintained by the sympathetic (accelerator) and vagus nerves at the pacemaker.

A tachycardia which is produced by lowered vagus tone at the pacemaker is called a *simple tachycardia.* Although the rate may be high, it is subject to the same influences that are normally exhibited in controlling heart rate. The acceleration may take place quickly but it is not entirely abrupt. The rapid rate may show further variations while maintained at a high level. The return of the rate to its usual level is rather gradual.

Another type of tachycardia is not infrequently met with. Because of its clinical manifestations it is called *paroxysmal tachycardia.* The exact mechanism of its origin is not fully understood, although its clinical and electrocardiographic features are well known. It is not under vagus control and its rate, therefore, does not change with vagus influence. There is some evidence that it is due to some sort of circus movement in a limited area of muscle. The focus from which such an ectopic rhythm originates may be in the auricle, in the ventricle or in the a-v connecting tissues. Most commonly it is supraventricular. The ectopic focus abruptly assumes the rôle of pacemaker and as suddenly yields again its new rôle.

There is this relationship between premature contractions and paroxysms of tachycardia: both originate in an ectopic focus which elaborates stimuli at an abnormally rapid rate. Patients who are subject to paroxysms of tachycardia frequently exhibit, during the interval between attacks, premature contractions whose electrocardiographic form indicates that they arise in the same focus whence the tachycardia originates.

Fig. 335 is an electrocardiogram from a case of paroxysmal tachycardia with origin in the auricle. With each cycle the

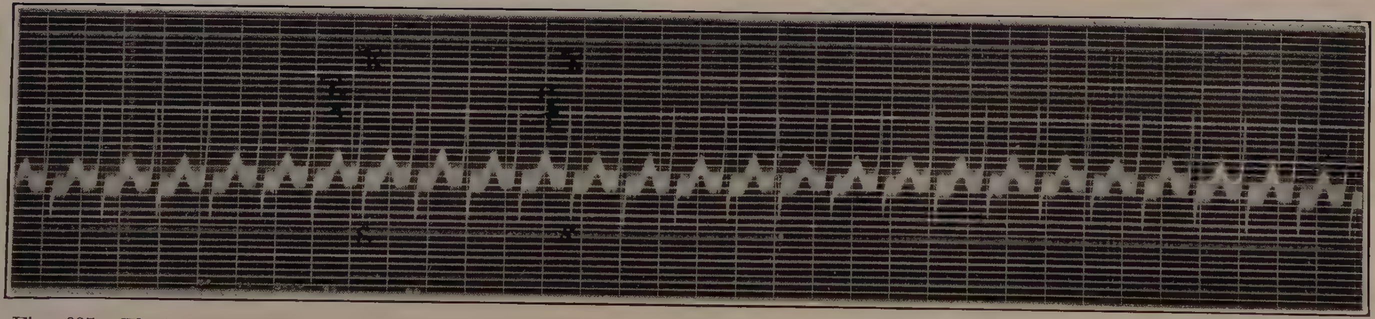

Fig. 335.—Electrocardiogram of paroxysmal tachycardia with origin in the auricle. Ventricular complexes are of supraventricular form. *P* and *T* waves are superimposed. Rate 222.

auricular wave P is superimposed on the T wave of the preceding ventricular complex and both are seen as one broad upward deflection. The ventricular complexes are of supraventricular type.

Fig. 336 shows two electrocardiograms each of which portrays paroxysmal tachycardia of another kind. In the bottom strip is recorded a short paroxysm in its entirety. The complexes are of ventricular type. Their shape is similar to that of the single ectopic beat near the right end of the figure. The rate of the ectopic rhythm is about 215.

The top strip is also a record of ectopic tachycardia of ventricular origin. The ventricular complexes are of abnormal type. The auricular waves are not well shown, being superimposed on the ventricular waves. Traces of them are clearly seen, however, as modifications of the ventricular waves. They are lettered P. The ventricular rate is about 220, the rate of the auricle about 115.

The Signs and Symptoms of Paroxysmal Tachycardia.—The attacks or "paroxysms" of tachycardia begin and end abruptly. This is perhaps the most characteristic feature. The termination may be marked by the occurrence of a few irregularly spaced beats before the normal rhythm is assumed. Except in the case of paroxysms of quite brief duration, the patient is conscious of the onset and ending of the attack, and while it lasts he feels the pounding of the heart. The rate during an attack is remarkably constant and is not influenced by posture, exercise, etc. It is usually found to be between 140 and 190, though rates above and below those figures are observed.

The paroxysm may be brought on by exertion, emotion or other influence or it may occur spontaneously. The duration is variable. It may last for only a few beats, or it may persist for days. Most commonly it is a matter of several hours.

Paroxysms of brief duration usually cause few symptoms other than a consciousness of rapid and forceful heart action and usually a sense of weakness. With longer attacks, however, more serious consequences usually follow. The feeling of weakness and faintness increases. Gastric symptoms often ensue. It can sometimes be demonstrated that the heart has dilated to some extent. The blood pressure falls. The heart sounds, which have assumed a tic tac quality, may become fainter with the longer duration of the attack. Congestion and edema of the lungs with frothy, bloody sputum, and other signs of failing heart may ensue. In most instances the attack has subsided before these more serious

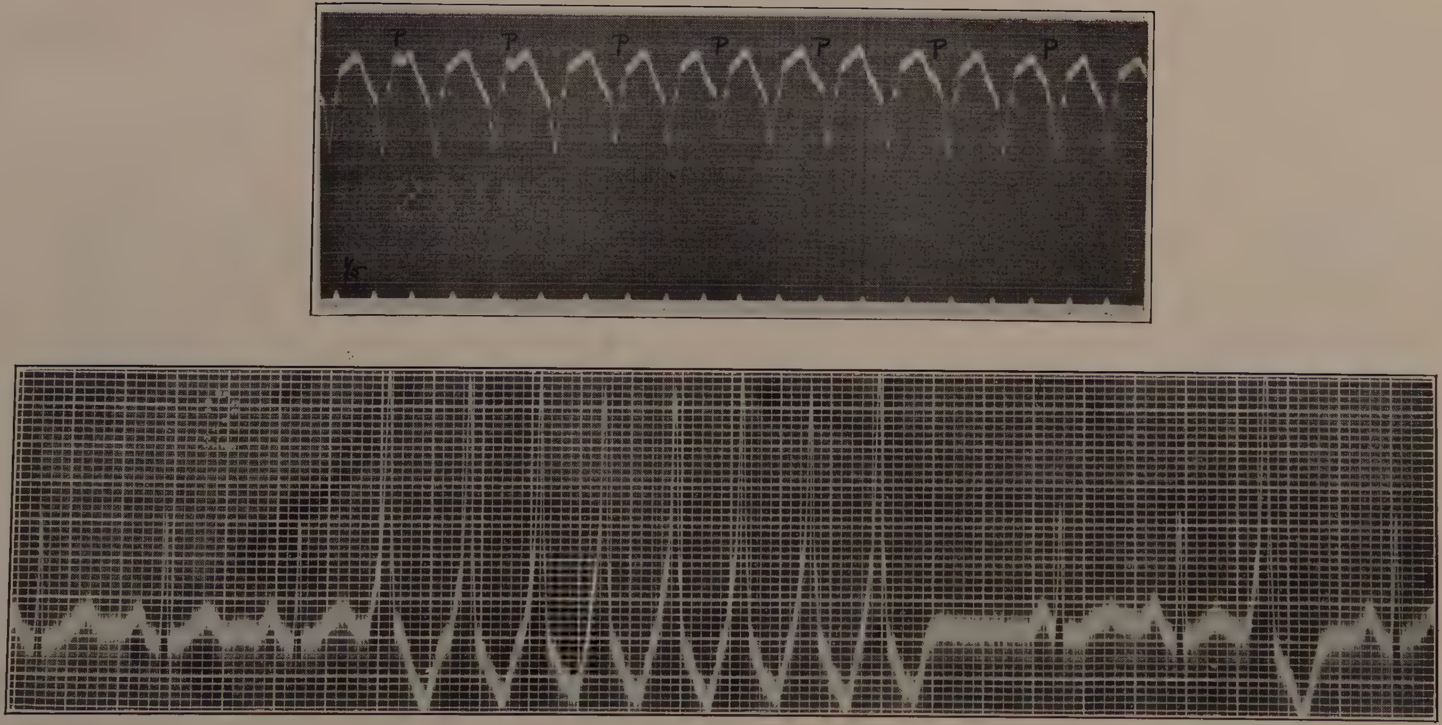

Fig. 336.—Two electrocardiograms in Lead II each showing ventricular tachycardia. The first shows a part of a long paroxysm. The second records a brief paroxysm. In it is recorded also an isolated extra systole. Complexes are of ventricular type. In the first record traces of auricular waves are seen at points lettered *P*.

symptoms make their appearance. The patient then experiences a sense of relief and rapidly recovers from the symptoms produced by the paroxysm.

There is usually a sense of oppression in the chest, and not unusually anginal pains occur, especially in the longer paroxysms. Death is a relatively infrequent ending of attacks, but is not altogether uncommon with those of long duration.

Many subjects of paroxysmal tachycardia learn certain measures which cut short the attack with more or less consistency. One may cause a cessation by taking and holding a deep breath, and especially by raising the diaphragm with the glottis closed after a full inspiration. Another makes strong pressure in the epigastrium, another bends forward and downward; thus producing the same effect. Other measures include the taking of gastric irritants and the induction of emesis. In paroxysms of auricular origin pressure over the vagus often stops the attack. It does not slow the rate. Either it stops the paroxysm or it has no effect.

Levine has called attention to other observations which may enable the examiner under favorable circumstances to differentiate ventricular from auricular paroxysms without graphic records. In the case of a ventricular paroxysm very slight irregularities occur even though the minute rate remains constant. Independent beating of the auricle may occasionally reinforce the first heart sound by the occurrence of auricular systole simultaneously with ventricular systole, thus producing a variation in the intensity of the first heart sounds.

ALTERNATION

(Pulsus Alternans)

The contractions of the ventricle under normal conditions are of equal force. Certain hearts, however, which are accomplishing their work with difficulty, exhibit an alternate variation in the strength of individual beats while maintaining a regular rhythm. This occurs particularly in cases of sclerosis and high tension which give other signs of failing myocardium. The ventricle expels alternately variable quantities of blood. The exact mechanism responsible for this phenomenon has not been fully explained.

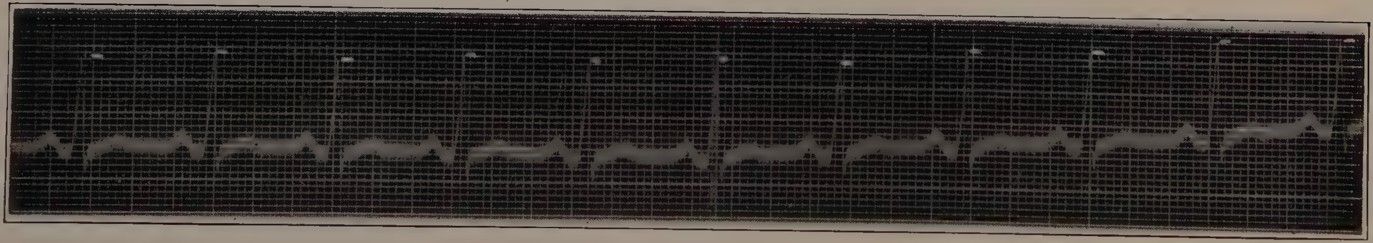

Fig. 337.—Electrocardiogram showing alternation. Short horizontal lines are drawn at the level of the peak of each *R* wave. Note the alternate variation in the height of the *R* waves.

Clinical Signs.—The difference in force may be great enough to be appreciated upon feeling the pulse. By careful palpation the alternate variation can often be detected in this way even though the difference is slight. The diagnosis may be exactly made by applying the sleeve of a sphygmomanometer to the arm and noting that there is one level of pressure at which the weaker beats disappear and a higher one for the alternate beats. By thus applying the correct pressure the arterial rate may be exactly halved.

Occasionally the difference in force is so great that the pulse rate is halved, the smaller beats not being perceived. This, however, is quite rare. The heart sounds may exhibit no difference in quality. A pulse tracing shows the alternation in the height of the arterial waves. In some cases the pulse tracing shows also an alternation of the intervals between beats, the smaller beat coming after the longer interval. This variation in *pulse rhythm* may occur even though the heart is beating rhythmically. By careful examination such a condition should be differentiated from regularly recurring extra systoles. In the latter the weak beat occurs after the short interval.

The electrocardiogram may give no indication of alternation. In some cases it shows an alternate variation in the height of the *R* waves.

Fig. 337 is an electrocardiogram in which the alternation is depicted. There is a slight, though definite, alternation in the height of the *R* waves. To insure clearer reproduction in the text, the peaks of the *R* waves in this figure have been indicated by short horizontal lines.

DIAGNOSTIC THESES

In attempting to diagnose abnormalities of cardiac mechanism without instrumental means certain generalizations should be kept in mind.

It is of value to examine both heart and pulse: of these more may be learned from the heart.

When the heart action is irregular it is important to determine whether there is a *predominant rhythm;* i.e., a rhythm which, irrespective of interruptions, dominates the action as a whole. If no such predominant rhythm can be demonstrated, the arrhythmia is probably due to auricular fibrillation. This probability increases if the rate is high.

It should be determined whether or not there is a *pulse deficit.* Neither occasional heart block nor sinus arrhythmia produces this phenomenon. Auricular fibrillation usually does so, provided the rate is high. A pulse deficit is common with extra systoles. It is rare with alternation.

In doubtful cases with slow rate an attempt should be made to accelerate the action of the ventricle. If the arrhythmia disappears with higher rate it is not due to auricular fibrillation; if it becomes more pronounced the probability of fibrillation is increased.

Occasional interruptions to an established rhythm are probably due to premature contractions. A regularly recurring interruption is probably due to premature contractions or to partial heart block, more likely to the former.

A bigeminal pulse may be produced by premature contractions which recur regularly after every second beat and fail to reach the wrist, or by the occurrence of a premature contraction which reaches the wrist after every normal beat. It may be produced by 3:2 heart-block.

An exact halving of an observed pulse rate may be produced as follows: (a) by the occurrence after every normal beat of a ventricular premature contraction too weak to reach the wrist; (b) by the onset of 2:1 heart-block; (c) by the change from a 2:1 auricular flutter to a 4:1 type; (d) by the onset of pulsus alternans of extreme degree.

A regular ventricular action which remains of constant rate from day to day, and which is uninfluenced by exercise and posture is probably due to flutter or to paroxysmal tachycardia. If the rate is less than 100 it is almost surely flutter.

Vagus pressure is often a useful procedure in differential diagnosis. Sinus arrhythmia usually is accentuated by it. In auricular flutter it usually produces an abrupt and conspicuous slowing for a few cycles. Occasionally it produces slowing in auricular fibrillation but usually has no effect. It frequently abolishes paroxysms of auricular tachycardia but does not abolish ventricular paroxysms.

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